SECOND SERIES APRIL 1943 VOL. 19, NO. 4

# BULLETIN OF THE NEW YORK ACADEMY OF MEDICINE

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*Published Monthly by* THE NEW YORK ACADEMY OF MEDICINE
*2 East 103 Street, New York*

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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

APRIL, 1943

# THE PRESENT STATUS OF SHOCK THERAPY OF MENTAL DISORDERS*

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THE task of evaluating the situation indicated by the title of this paper is not an easy one, and the selection and presentation of the facts now available constitute difficulties which are not satisfactorily surmounted. On the one hand a statistical presentation composed of the results accumulated from the great variety of viewpoints and experiences in vogue would be far from enlightening since statistical treatment of doubtful material is seldom if ever helpful; while on the other hand a comprehensive, detailed description including all that is pertinent in the now-extensive field of shock therapy would be too extensive to present in a short treatise.

For the past five or six years the pharmacological shock therapies have dominated psychiatric research with their main focus in schizophrenic disorders. They have also infiltrated other branches of medicine. During the year 1941 and what has passed of 1942, some one hundred fifty-seven published articles on the action of insulin, metrazol, electric

* Presented October 21, 1942 at the fifteenth Graduate Fortnight of The New York Academy of Medicine.

currents, and other shock producers, have appeared in the scientific literature of the world. Of these fifty dealt with the effects of the convulsant, metrazol, forty-eight with electroshock methods, sixteen with insulin, and forty-three with other coma-producing drugs and procedures (nitrogen, sodium amytal, and combinations, etc.) Only a few of the most recent pertinent contributions can be mentioned in this paper.

Although this is sufficient evidence to show that a great deal of work has been done and is still going on with the shock methods, these published reports present a discordance in the results obtained. There is as yet no consensus on the basic value of any of these treatments, some authors being very enthusiastic over their results, others being unconvinced of favorable results, and still others expressing frank opinions on the inadequacy and even harmfulness of such procedures. One thing is certain, shock therapy is receiving an extensive clinical trial as it has been widely adopted in mental hospitals of all kinds, in general hospitals, in out-patient departments and clinics, and in private offices of general practitioners, as well as in those of psychiatrists.

There is a sufficient skepticism among this horde of workers to insure a careful investigation and testing of the numerous problems and questions which have been raised and to promise some more reliable information about several all-important items. Experienced psychiatrists are concerned with the following questions on which there are positive opinions supporting both sides based on reported experiences:

1. Will shock treatment finally go the way of many, if not most, other "cures" which have had their period of popularity and enthusiastic support only to be discarded as not worth pursuing further? The history of the treatment of schizophrenia illustrates particularly the significance of this question.

2. Is this treatment a specific? Does this radical approach offer any lasting benefit? This question recurs in the minds of those psychiatrists who note that patients go from one hospital or sanitarium to another receiving different kinds, degrees, and "courses" of shock therapy without any tangible lasting results. Are these patients exceptions or are relapses always to be expected?

3. Is the treatment being applied improperly, inadequately, or dangerously by many hospital staffs and private practitioners?

4. Does it aid the patient to understand his personal problems and

does it help the psychiatrist to understand the patient, his disorder, his problems, or his constitutional equipment?

5. Does it tend to discourage organized beneficial psychotherapy in general and to encourage neglect of the same in individual patients in particular?

6. Which type of shock therapy is the most efficient and under what circumstances, and for what disorders is a special therapy selected? Is electric shock the most efficient in certain disorders or is its growing popularity and preference based on its relative simplicity of application, its after-care advantages, and its inexpensive features as compared to insulin, for example?

7. Does the danger of damage to the body structures, and to the brain in particular, outweigh any benefits that might be obtained by any of these treatments?

While there is as yet no satisfactory answer to many of these questions, some comments regarding them based on reports, experiences, and experiments are in order. Discordances in results and findings in any scientific work indicate the necessity of additional investigation and suggest that there is some lack of uniformity in the selection of material, in the technical procedure, or in the interpretation of the results, particularly as to what constitute "cures," recoveries, remissions, improved, and so forth. It is rather obvious that this is the case with shock therapy.

Regarding the indications for treatment and the selection of patient material for the application of shock methods, some believe that in order to test the efficacy of the treatment patients should be selected for whom the prognosis seems to be hopeless, and who have resisted all other forms of therapy; others have treated everything from character disturbances and mild neuroses to severe psychoses with organic brain diseases, not omitting a try at general paresis.[1]

It should be emphasized that all of our classifications of knowledge, including those of mental disorder, are constructed by man out of his impressions and are not necessarily made so by nature, therefore they may not be valid and are probably at best only temporary in significance. There is no distinction in nature between "organic" and "functional," a concept which is man-made and probably does not actually exist biologically. As far as our knowledge of correlations is concerned, there seems to be a greater gap between "anatomical" and the "physiological" than between the "physiological" and "psychological." The

TABLE I

COMPARATIVE RESULTS WITH DIFFERENT METHODS

| *Insulin* | *Metrazol* | *Electroshock* |
|---|---|---|
| 1. Catatonic dementia precox | Involutional melancholia | Involutional melancholia |
| 2. Paranoid dementia precox | Catatonic dementia precox | Manic - depressive psychosis (depressed phase) |
| 3. Hebephrenic dementia precox | Manic - depressive psychosis (depression) | Catatonic dementia precox |
| 4. Involutional melancholia | Other depressions | Manic - depressive psychosis (manic phase) |
| 5. Manic - depressive psychosis (depressive phase) | Manic - depressive psychosis (manic phase) | Other depressions |
| 6. Manic - depressive psychosis (manic phase) | Psychoneuroses | Psychoneuroses |
| 7. Psychoneuroses | Paranoid dementia precox | Paranoid dementia precox |
| 8. Simple dementia precox | Hebephrenic dementia precox | Hebephrenic dementia precox |
| 9. Alcoholism | | |

manner in which structure performs its physiological functions is not more thoroughly understood than is the physiological-psychological relationship.

Regardless of the uncertainty and lack of uniformity in diagnostic schemes and classifications, an analysis of the literature and Kolb's[2] (1942) comprehensive survey of 305 mental hospitals show a certain amount of consensus on what types of cases may be treated with a preferred shock method, with some expectation of success. The indications in diagnostic terms, and in order of the frequency of success with the three main shock therapies, are presented in Table I.

Combinations of insulin and metrazol, and of insulin and electroshock, are used and are given preference by many workers, but speaking generally the results have created a choice of insulin for dementia precox (or schizophrenia), and the convulsion-producing methods, metrazol and electric current, for involutional and other depressions, including those in the manic-depressive syndrome.

Psychoses called "dementia precox" and "schizophrenia" have received a great deal of attention and may serve as examples demonstrat-

ing some of the reasons for a lack of uniformity in therapeutic results. It was long considered erroneously as a hopeless disease. The large number of new cases developing each year seems to have obscured the fact that remissions were not infrequent before the era of shock therapy. According to reports, Sakel's insulin method has produced as high as 70 per cent remissions.

At practically all psychiatric clinics the diagnosis of "schizophrenia" is made as a collective affair which includes a number of clinical pictures so atypical as to suggest an entirely different genesis. These atypical or pseudoschizophrenic forms compose the majority of "schizophrenic" patients treated in some clinics. Herein may lie the difference in reported clinical results which are certainly more favorable in the pseudoschizophrenic forms than in the genuine nuclear types, and they cannot serve to test the value of any method of treatment to be interpreted in terms of the whole category of schizophrenia. Clinically they may resemble atypical depressive and manic states, or show alternating conditions of excitement and stupor with affective trends, or have projection symptoms (often called "paranoid") that are compensatory but not deeply seated and need not be nuclear in nature. In studying the effect of therapy, it would be highly desirable and advantageous to separate these conditions from the main group and designate them as atypical schizophrenic reactions.

Whatever it is that releases a latent schizophrenia in an individual, whether it be in an endocrine setting, a metabolic dysfunction, an exogenic intoxication, an infection, or a psychogenic trouble, it seems logical that the character of the onset, the nature of the symptoms, and the final result depend upon both the qualitative and quantitative relationship between the product of the chromosomal components and the precipitating factors. Studies of the individual over a number of years, which include the prepsychotic character, the intellectual status, the social and physical disease factors, the bodily form, and the onset and course of the psychosis have given us some pertinent information on what varieties of reaction tend generally toward recovery, remission and improvement, and those which tend in the direction of a poor prognosis regardless of the type of treatment applied, if any, of a special nature.

Bleuler, who was a master observer in this field, found that schizophrenia in which catatonic features developed gradually offers a bad

prognosis; moreover it is our experience that cases of typical catatonia without paranoid components or other mixtures have an unfavorable prognosis and retain most of the expressions of the disorder throughout the course, a fact which we interpret as characteristic of an organic disease, systemic in nature. Schizophrenic psychoses comparatively poor and colorless in symptoms have an unfavorable prognosis unless the person is of a low order of intelligence in which case the prognosis is much better. Kretschmer emphasized that the asthenic body form is not only highly predisposed to schizophrenia but also carries a poor prognosis regardless of the type of therapy employed. However, remissions do sometimes occur in those with typically asthenic habitus and schizoid personality. Schizophrenia precipitated by psychic trauma yields an unfavorable prognosis if there is a prepsychotic personality characterized by stubbornness, inelasticity, and constriction of interests; however, if the trauma occurs to one with a rich personality with many interests, recovery is common.

In contradistinction to the varieties just mentioned those patients, whose psychoses begin with an atypical acute phase, or who have a pyknic habitus, a cyclothymic temperament, or prominent exogenic causes, or some combination of these, have a definitely favorable prognosis. Among the unfavorable outcomes the pyknic habitus has been more rare, while cyclothymic temperaments are more frequent; therefore they do not always occur together, as is sometimes assumed. Of the two the pyknic habitus seems to count for more than the cyclothymic temperament in evaluating the prognosis. Additional research may reveal which is of the greater significance for the prognosis, the body constitution or the kind of temperament. A combination of pyknic habitus with cyclothymic temperament and depressive features in the symptomatology of the acute phases of the psychosis is often present in the remitting cases, also exogenic traumas, or precipitating factors prior to the onset of the disorder, have been relatively numerous. Among these have been alcoholic abuses, thwarted love affairs, unemployment conflicts, operations, head traumas, family conflicts, and childbirth.

In some instances after "improvement" or "social recovery," the mental status of the patient remains the same for several years showing only slight or perhaps concealed oddities and mild disorders of thinking. He may retain a number of the symptoms of autism and constric-

tion of ideas, but these have become isolated, influencing his daily behavior very little. He has adjusted himself to the world with personality well preserved and capable of making his living. He is usually classified as "cured" and may be so considered by those about him.

All of these natural and sometimes spontaneous reactions toward improvement and recovery, with their compensatory features, must be kept in mind while evaluating the results of shock therapy. Shock therapy may and apparently does exert a favorable effect upon this tendency to get well, and it has been the experience of most workers with these methods that hallucinations, mild paranoid ideas, and borderline symptoms tend to disappear, at least temporarily.

Other sources of discord in estimating end results are the time allowed to elapse between the end of the therapy and the final examination; and the lack of much-needed follow-up studies. Some results are reported immediately after the completion of the treatment while others report at various post-treatment intervals.

Some valuable, careful follow-up studies were made by Bond[3] on 125 insulin-treated schizophrenic patients and 153 consecutive schizophrenic patients for controls at the Institute of the Pennsylvania Hospital in Philadelphia (1941). Here the use of insulin has been followed by immediate and important gains in one-half of all patients, and in about two-thirds of the acute cases of less than eighteen months' duration. After two years these gains were maintained in only one-fourth of all patients, and in about one-half of the acute cases. Similar gains were noted in one-sixth of the control cases, two years from the time of admission and without relation to the previous duration of the illness. Bond's experience indicated that while insulin shock is strikingly effective in schizophrenia, there is need for supportive measures to hold these gains which appear quickly but tend to disappear. Such studies are necessary and are long overdue from many clinics where the treatment has now been used for several years.

Cheney and Clow[4] in 1941 reviewed results in 50 cases of dementia precox from six months to two and one-half years after insulin, thus gaining some impressions of prognostic value. According to these authors, the most favorable outlook is in men under 30 years of age who have the history of a comparatively adequate prepsychotic personality, whose psychosis had an abrupt onset with a definite external precipitating cause, an illness of less than one year's duration, and the sympto-

matology of an excited catatonic state without evidence of deterioration. Those least likely to benefit are women over 32 years of age with adequate personalities, insidious onset, no definite external precipitating causes, who develop mixed clinical forms with evidence of deterioration and with the disorder usually of more than one year's duration. Various combinations of these prognostic factors occurred in different patients, but their relative predominance influenced the outcome. Patients who were to improve usually showed this relatively early in the course of treatments. If the results did not appear early a prolonged course of treatment rarely produced recovery. Previous attacks with remissions did not seem to influence the outcome.

Patients who will benefit by insulin therapy have the same characteristics as those formerly improved by other forms of treatment. This may indicate that insulin has no specific curative effect but accelerates or facilitates improvement in those who have the constitutional or innate capacity for improvement. Cheney and Clow believe that it should be supplemented by psychotherapy and occupational therapy. In their series, the results indicated that a 16 per cent recovery rate may be expected after intensive treatment with about one-half of those to remain benefited after 6 months to 2½ years. The treatment if confined to favorable cases, however, may result in an improvement rate of 90 per cent at the end of the insulin course, and of 80 per cent at the end of 6 months to 2½ years.

The results to be expected and the degree of extension of the therapy justified will depend eventually upon an accumulation of follow-up and comparative studies of this character. The more recently developed electric shock method is now in the front ranks for preference in treating certain mental states and our comments on the present clinical aspects must be brief and center around a few of the most important features.

A recent (1942) report by Hemphill[5] on his experiences with 200 cases can be used as a sample investigation. This series contained 137 women and 63 men, and I have taken the liberty to arrange his results in Table II, using his own diagnostic terminology:

Thus the results were highly satisfactory in the depressives and manics, i.e., the affective disorders, and the author emphasized this particularly for involutional melancholia which was included with the "depressives"; but results were very poor for the schizophrenics, the chronic

TABLE II

| | *Recovered* | *Improved* | *Improved in Hospital* | *Unimproved* |
|---|---|---|---|---|
| Depressives | 35 | 6 | 0 | 4 |
| Manics | 14 | 2 | 2 | 1 |
| Schizophrenics | 4 | 8 | 26 | 76 |
| "Mixed" psychoses | 4 | 3 | 0 | 4 |
| Psychoneuroses | 2 | 4 | 1 | 4 |
| | 59 | 23 | 29 | 89 |

patients showing social recovery rather than a more complete restitution. The effect was a little better in the more recently developed cases. Hemphill in generalizing stated that the best results were obtained by three convulsions weekly, and that the recoverable cases usually showed some improvement before the fifth fit. He warned that physical illness, sepsis, and prophylactic inoculations within 48 hours of treatments are contraindications, and also there is a possibility of reactivating pulmonary tuberculosis.

It has been noted that in Hemphill's report poor results were obtained with his group of patients diagnosed as "schizophrenics." In contrast with these poor results one should mention the investigation of Kalinowsky and Worthing* who found that electric shock alone may be of great benefit in the production of remissions in early cases of schizophrenia and provided that the series of convulsions is of sufficient length. When broken down the material illustrates emphatically the importance of early treatment in schizophrenia, and on the basis of our personal knowledge of this work one should state that the sufficiently long series of treatments is of great importance. The results of these authors also show that as the duration of the illness increases a much smaller percentage of remissions occur.

Impastato and Almansi[6] (1942) among others have insisted that the patients should be studied carefully neurologically, roentgenologically, and biochemically, in addition to having electrocardiographic and elec-

* *Psychiatric Quarterly*, 1943, *17*:144.

troencephalographic evaluations in connection with the treatment. They refer to the impression, however, that the anxiety and tension due to the mental disorder may be worse physically for the patient than some of the supposed "contraindications." For example, in diseased heart conditions the convulsion may be less damaging than the constant tension due to anxiety and depression. At any rate, cardiac patients have been treated successfully and so have those with hypertension, as well as those who have reached the 70 year level or above with circulatory systems showing evidence of senility and arteriosclerosis.

Clinically there are several interesting points among which are the instantaneous relaxation, the reduction of all external stimuli, the lessening of internal tension characteristic of several mental disorders, and the amnesias that follow the seizures which persist for some time in various degrees. These seem to operate therapeutically with the amnesia aiding the patients to forget their emotional problems temporarily and thus eventually breaking up the psychopathic pattern. In the successful case the patient has gained a more complete control over the dominating psychopathologic ideas, and the immediately favorable results are startling, even miraculous at times in the involutional melancholias, agitations and other depressions.

In general the results of electric shock therapy may be little if at all superior to those reported following metrazol, but there is a growing preference for the former method. Some of the reasons are: (1) the technique of application is much simpler and there is no problem of inaccessible or thrombosed veins; (2) a convulsion is practically always produced which is less violent and of shorter duration; should the fit fail to develop there is seldom excitement; there is rarely any post-convulsive excitement and fewer post-treatment convulsions have been reported; (3) unconsciousness always and usually amnesia for the treatment are produced; there is no pain, rarely disagreeable sensations, few or no unpleasant memories, rarely fear, and no feeling of dissolution and death which is apt to appear in the metrazol picture; and (4) complications are less common; it is less dangerous in organic conditions; there are fewer vertebral compressions; and the death rate is not as high as that reported for metrazol.

The method is less expensive and more easily applied than the other shock therapies, and perhaps herein lies the danger. It is actually a rigorous, major procedure following which unexpected things sometimes

occur, such as panic over the memory disturbance which, by the way, may remain over a long period in some cases, confusional episodes, and excited states. For these reasons and other possible complications it is desirable to carry out the therapy under the protection of a hospital environment, in fact the physician who does it outside the hospital or without an equivalent protection for the patient is assuming unnecessary, as well as unwise, responsibilities.

The most commonly mentioned complications of a physical nature following shock therapy are cardiac and other circulatory disorders including cerebral vascular accidents, pneumonia (aspiration and bacterial), activation of tuberculosis, lung abscess, fractures and dislocations, sore back, and memory defects. This array of possibilities should convince anyone that we are dealing with a radical form of therapy and one that should be as carefully applied and controlled as possible. Occasionally death results either directly or indirectly. Kinsey[7] (1941) reporting on all published cases found the incidence of death to be 73 per 10,000 patients treated with insulin, and 23 per 10,000 treated with metrazol. Classifying these deaths under cerebral, cardiac, and pulmonary causes, the order of frequency was as follows:

*Cerebral* —Hypoglycemic encephalitis (insulin) (38 of 90 deaths)
Status epilepticus
Hemorrhage and edema
Embolism (metrazol)

*Cardiac*

*Pulmonary*—Aspiration pneumonia
Other pneumonias
Abscess of lung
Tuberculosis

As the brain is the most sensitive structure of the body, one wonders whether the application of these powerful measures does not result in some irreparable damage. There are a number of histopathological investigations bearing on this question from both human and experimental animal material. In human brains from those dying after insulin therapy, Ferraro[8] (1942) found zones of rarefaction in various cortical areas due apparently to the gradual disappearance of affected nerve cells, as well as the absence of cells in patchy, focal areas where they had undergone degeneration and disintegration. There were areas of cortical devastation involving various layers with disturbed cortical cy-

toarchitecture particularly in the frontal and temporal areas and in the purkinjian layer of the cerebellum.

The so-called "toxic" reaction characterized by acute swelling and chromatolysis, which is reversible to some extent, is common. There are also ischemic changes in the cells in the region of the blood vessels. Fatty degeneration of the neuron is common and there is an increase in intracellular lipoids particularly in the temporal lobes according to Kobler[9] (1938), Kastein[10] (1938), Terbrüggen[11] (1931), Ferraro and Jervis[12] (1939). There is some question as to whether this is a non-specific reaction in toxemia or the result of a specific action of insulin upon the intracellular metabolism of lipoids. Rivore, Staub, and Asher have pointed out that sugar under the action of insulin may release fatty substances in the tissues. Kobler[9] and Malamud[13] (1938) maintained that insulin probably has a damaging effect upon nerve cells, and Holmes[14] (1930) thought that insulin diminishes intracellular oxidation in the brain.

The blood vessel changes were described at length by Ferraro[8] (1942). They are productive in nature consisting of proliferating changes of the intima, and hyperplasia and swelling of the intimal endothelial cells. This vascular proliferation may be related directly to insulin intoxication as the change is similar to that reported numerous times by investigators as due to various other toxins such as lead, organismal toxemias, and infections.

At Northwestern University, Weil and Liebert[15] (1940) studied the brains from six mental patients who died 2 to 10 months after treatment with metrazol injections. The outstanding hospitalization features were marked hypertrophy and hyperplasia of astrocytes, and, to a lesser degree, the microglia. Disease of the ganglion cells was less pronounced. In the case of more recent illness (two to three years' duration), the reaction was more pronounced than in those of five to ten years' standing.

Arieti[16] (1941) at the New York State Psychiatric Institute made histopathologic observations on 5 monkeys after metrazol-induced convulsions. Histopathologic changes were not always present in the central nervous system. Moreover when they did occur they were not proportional to the number of convulsions, dosage of the drug, or the duration of the seizures. The alterations were mild, more so than those described as due to insulin treatment. There were usually small groups

of neuron cells showing acute degenerative changes, but some revealed the severe type of degeneration described long ago by Nissl. Anemia of the external cortical layers, anemic and hyperemic foci in the inner areas, and general breaking up of the network of capillaries of the brain were also noted. Interstitial and parenchymatous involvement of the kidneys were always found.

Regarding the action of the electric shock, Löwenbach and Stainbrook[17] (1942) state: "A generalized convulsion leaves a human being in a state in which all that is called the personality has been extinguished. Although the autonomous functions are operating more or less regularly, the individual does not react for a while to any kind of stimuli and the activity of the cerebral cortex as revealed by the electroencephalogram has completely ceased. The convulsion is succeeded by a phase during which the higher functions return and in which the personality is reintegrated. It seems as if the subconvulsive reaction does not differ from the convulsive except in degree, but in these minor reactions where the brain has not been completely exhausted reintegration may begin at any level, and the recovery may be much more quickly and indeed sometimes imperceptibly achieved."

The extensive use of electric convulsion treatment in the psychoses makes it imperative to ascertain what happens to the brain during or after such applications. It is a matter of great practical importance that we gain some knowledge of what is responsible for the confusional state and the amnesia following the treatments, and particularly as to whether these states are due to some form of structural damage. If the electric current has damaged the brain permanently there have been but few clinically demonstrable ill effects when the specified doses and the proper adjustments have been carefully followed, but this form of treatment should not be used without the utmost discrimination and caution until more is known about the possible danger to the nervous system.

Alpers and Hughes[18] (1942) studied two cases treated by electroshock. One patient dying after 62 convulsive treatments revealed fresh hemorrhages in the cerebral cortex, white matter, and brain. The brain of the other patient who died five months after the final treatment showed old areas of perivascular damage mainly in the white matter. These lesions seemed to be due to the convulsions and the question was proposed as to whether these changes might persist indefinitely. The perivascular areas in some parts of the brain may be capable of produc-

ing some types of sequelae.

Subarachnoid and punctate hemorrhages in the brains of cats subjected to electrical convulsions were described by the same authors[19] (1942); no lesions had developed in the pons and medulla. Similar findings were reported by Heilbrunn and Weil[20] who treated rabbits and rats in keeping with the procedures used with humans. No generalized ganglion cell lesions or generalized proliferative glial reactions were observed but there were changes in the venous system in practically all cases (25 out of 28). Hemorrhages were present in the meninges and in the substance of the brain and spinal cord. These hemorrhages were confined to the perivascular regions of the capillaries, being caused by rupture of the walls.

Changes in the electrical activity of the brain as demonstrated by Pacella, Barrera, and Kalinowsky[21] (1942) indicate that there is an immediate change in the cerebral physiology somewhere, but the nature and degree of any permanent pathological change have not been definitely established. An insufficient number of cases have been examined pathologically and as far as the human material is concerned any examination of the brains from those dying during or after the treatment must differentiate as to which lesions, if found, are due directly to the action of the therapeutic measure, which are caused indirectly by various somatic-physiochemical processes released by the therapy, and finally which are produced by other processes and bodily lesions playing a major role in the lethal event. The brain must be examined in the light of all other postmortem findings and conditions in the body before any final evaluation can be made in a given case, as the brain participates in many lethal conditions the foci of which lie in the other organs of the body.

## Conclusions

1. Insulin, metrazol, and electroshock with the adjuncts that have been recommended by some workers are all valuable in proper hands and particularly when they are supplemented by systematic psychotherapy and other measures available in psychiatric clinics. In some instances they have undoubtedly been misused. They are not a panacea for all mental disorders. Superintendents of mental hospitals and practicing physicians have had numerous requests to apply these treatments to the mentally defective, the aged, deteriorated patients, and others

entirely unsuitable in every respect. Such requests, often leading to unpleasant controversies, are due usually to the false hopes aroused in relatives and friends by startling statements in the press and the inaccurate reports in the numerous recent books on psychiatry written by enthusiastic laymen who, with all good intentions, peddle misinformation.

2. At the present time there is no theory as to the nature of the shock reactions sufficiently comprehensive to be taken very seriously. Theories not susceptible to experimental examination are hardly worth discussing. There is as yet insufficient knowledge to justify attempts in the direction of theory building. Serious efforts to sort out existing knowledge with the expectation of making some sound deductions may be made so that in time a comprehensible formulation of the fundamental elements in the problem will be possible.

3. If statistics on insulin therapy are taken as indicative of therapeutic results in schizophrenia, every possible stated percentage of "cures" can be found from Sakel's 70 per cent to Langfeldt's experience of "no cures" in the clear-cut nuclear cases. It is probable that 70 per cent of satisfactory remissions would not be unusual in those clinies where the material is composed mainly of the atypical varieties of schizophrenia. When the diagnosis is just "schizophrenia," results up to 60 per cent "recovered" or "improved" by means of any therapy do not indicate the relationship between that therapy and the disorder. This situation demands complete and accurate information, first, as to the form of schizophrenia, second, as to the special characteristics of the individual as a patient, and third, perhaps above all, as to the factors which will make possible the establishment of criteria for "recovered," "social recovery," "improved," "not improved," and so on, which will replace the present chaotic state of affairs in this respect on which statistics are prepared. Only precise statements and careful differentiation of cases can contribute toward bringing the problem to a point of national and international coöperation for solution. The same applies to mental disorders other than schizophrenia.

4. Possibly the shock methods should be used only as adjuncts in a total therapeutic approach, including psychotherapeutic interviews with the physician, physiotherapy, occupational therapy, physical education, and a program to promote socialization.

5. It is as yet too early to make any final pronouncement on the

results of electric shock therapy in the treatment of various mental disorders. Premature announcements of results may often be prejudicial to a proper evaluation of any method of therapy, either because of the utilization of an insufficient number of cases or because of selective factors entering into the determination of the types to be treated. Technical advances are also always under way to modify the picture. It is interesting to note that there are neurologists and psychiatrists who, without any extensive personal experience with any shock therapy, are nevertheless actively opposed to all shock therapies on theoretical grounds.

6. Electrically induced convulsion therapy appears to be a safe and convenient method of treatment in mental disorders. It is particularly satisfactory in the affective disorders and especially in "involutional melancholia," but it cannot as yet be regarded as a specific. Its value in chronic schizophrenic illnesses is more of the nature of promotion of social improvement than fundamental changes. Its effect on recent cases is much more favorable, and early treatment is well worth trying. Some workers have better results in schizophrenia when a comparatively large number of convulsions are given, the general tendency being to afford too few to promote a successful outcome.

7. So far there is no reason to emphasize any particular contraindication for electric shock therapy which does not already exist for any other form of shock treatment.

8. Combinations of the various types of shock therapy may be particularly valuable in certain cases as it does not follow that if one method, insulin, for example, fails to produce a favorable effect, some other method, such as metrazol or electric shock alone or in combination with insulin, will not produce good results.

9. The shock therapies are safe clinically if handled by individuals properly trained in their administration and in the handling of psychiatric patients. Mental confusions and other psychiatric emergencies will arise demanding expert attention and the psychiatric amateur may find himself at a serious disadvantage in such situations.

10. Although the shock therapy methods are not too securely established universally as entirely desirable procedures in treatment, there can be no doubt as to the stimulation they have given many fields of research including physics, biochemistry, pharmacology, physiology, pathology, psychology, and clinical medicine and psychiatry which

have produced a vast number of contributions. It can be said that the whole realm of psychiatry has been vitalized with a renewed interest that will lead to a better understanding of this complex subject and thus to progress in science.

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# THE SELECTIVE USE OF ELECTRO-SHOCK THERAPY AS AN ADJUVANT TO PSYCHOTHERAPY*

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I WOULD like to preface this paper by the emphatic declaration that I do not approve the indiscriminate use of electro-shock. Without doubt, it carries some risk which varies with the judgment, experience and discretion of the physician. As a therapeutic method, it has provoked repugnance in the minds of many psychiatrists because of its apparent violence; some have referred to it as "sadistic therapy." To this, I reply: "Yes, it appears to be a crude method, but it is the best we have available to us at the present time for the treatment of certain serious mental problems." I am convinced that lives have been saved by this method, lives that would have otherwise terminated in suicide. Moreover, it is a therapeutic method that often enables a patient to carry on the work necessary to his livelihood without the serious interruption of going to an institution. It has spared some people the stigma of having been confined in a mental institution; much as we may deplore the existence of such a stigma, it is a social reality.

We know, too, that the recovery of those patients whose condition requires them to be sent to the mental hospital or sanitarium can often be accelerated through shock-therapy. This is especially true of the melancholias. While less true of the patients with schizoid problems and the severe obsessive compulsive states who are seriously disabled by their torturing symptoms, there are instances in which even these mental disturbances have been favorably influenced by shock-therapy.

I should also like to state most emphatically that electro-shock therapy, like any shock therapy, is woefully incomplete without adequate psychotherapy. In my opinion, a grave injustice is done to any patient who does not receive intensive psychological assistance whenever pos-

* Read before the Section of Neurology and Psychiatry of The New York Academy of Medicine and the New York Neurological Society, November 10, 1942.

sible. By intensive, I do not mean one or two superficial interviews but a persistent investigation of those factors in the functioning of the personality which led to the mental breakdown. This therapeutic coördination assures a healthier individual, less likely to break down again in a critical situation.

Since it is presumed that practically everyone in this audience has become familiar with the technique of electro-shock therapy, I shall not go into technical detail extensively. The apparatus I used was a Rahm machine with the forceps electrodes; in nearly every instance, the current was for 0.10 second, varying between 70-120 volts. Most of the time, a grand mal convulsion was aimed at; occasionally, petit mal reactions were deliberately induced, but increasing experience seems to point toward the desirability of the major type of seizure to achieve maximum benefit.[1] This is particularly true with regard to the amnesia for the shock experience; the lighter the treatment, the less amnesia, and the less amnesia, the more disturbed the patient seemed to be after the treatment. This disturbance was not only immediate but would often be prolonged for one or more days.

Now, as to the theories regarding the benefits achieved by electro-shock therapy, I will mention only two. First, the physiological, and second, the psychological.

Briefly, the physiological interpretations for the improvement obtained by electro-shock are based upon the assumption and evidence that physico-chemical alterations occur. They are:

changes in oxygen-carbon dioxide ratio;[2]
changes in vascularity of brain areas;[3]
changes in the cellular structure of the cerebral cortex, as demonstrated by the electro-encephalogram;[4]
changes in blood pressure, velocity of blood flow, etc.;
changes in chemical contents of the blood.

Since it is relevant to the theme of this paper, I shall dwell more on the psychological aspects of the theories attempting to explain the improvement. There are a number of observers who believe that emotional shock based upon threat (of death) causes the change. Schilder had advocated this point of view and Berkwitz[5] postulates his technique of repeated daily faradic shock upon this threat factor, among other factors.

When we consider psychological dynamics in mental illness, we are

frequently struck with the patient's inability to do something about a situation. This state may reflect helplessness or inertia. It is my impression that this apathy or inertia is no inconsiderable barrier to the more adequate functioning of the personality and I believe that electro-shock produces the effect of jarring the individual so strenuously that he is summoned to the necessity of facing reality more directly and energetically.

My opinion is that the improvement which occurs is made possible by psycho-physiological alterations affecting *feeling tone*. By feeling tone, I refer to a quantum of energy tone relating to mood, quality of perception, attitude toward self and attitude towards the world, awareness of capacity to feel the ability to do things, the so-called *elan vital*. This would derive from those sources within the human organism which are energy producing, e.g., the glandular apparatus, especially the adrenal, the vascular apparatus, etc. This could also be related to the unconscious resistances of the patient to coöperate with those around him, to feel a willingness to help.

The question has often been raised quite naturally: "Does not this seemingly violent assault on the brain cause serious damage to the intellectual functioning of the electro-shocked individual?" It is part of my purpose to demonstrate that not only was there no perceptible impairment of intellectual integrity, beyond the immediate transitory confusional reaction, but that these patients seemed to do better than ever before! I realize this seems extravagant. When we bear in mind, however, the disabling effect of severe emotional disturbance on the intellectual coördination of an individual, we see that this is not so far fetched. The corrosive undermining of the thinking process which constant tension, anxiety, and resentment can cause, is familiar to every experienced student of the psychology of behavior. At this point I would like to stress again that increased assurance for such improved functioning is afforded by adequate psychotherapy to supplement the shock arm of therapeutic management. Such a combination is extraordinarily suitable to office practice because patients make their adjustment to their life situation as they proceed with treatment.

## Some Remarks on Dynamics of Therapy

The release of feeling that electro-convulsive therapy enables the patient to achieve is, in my opinion, a factor of importance in moving

the patient along the direction of recovery. Such feeling is perceptible to the trained observer during the initial period of behavior immediately after the seizure, whether of the grand or petit mal type. Particularly significant is the demonstration of hostile feelings: being resistive; gestures of threatening assault; or, being negativistic. Such acts as pushing, punching, slapping at persons surrounding the patient, glaring at them balefully, turning the back on questioning by doctors present are common. As mentioned above, these acts take on added significance when we recall that they tend to be repetitive and that the pattern is characteristic for the individual.

As criteria of progressive improvement, the following observations have been noted:

1. Raising of convulsive threshold (but not invariably).
2. Decrease in the violence of psychomotor phenomena
3. Increased rapidity of return to awareness of surroundings immediately following the convulsion
4. Lessening of memory impairment following convulsion in the later stage of treatment, compared to the early period
5. Usual clinical indications of improvement; being more cheerful; gain in weight; sleeping better; willingness to coöperate at home; more outgoing in behavior generally with a corresponding diminution of inertia; being more assertive to the extreme point of acting aggressively at times.[6]

It is of great interest to note the varying flexibility of patients in this release of feeling. In dealing with rigid personality structures where lack of spontaneous affective expression, impassivity of facial expression, and silence are pronounced, the array of postconvulsive psychomotor activity is relatively violent. This violence is in striking contrast to the daily behavior of the patient, particularly so in cases of depression. Then, as the spontaneity of feeling becomes more evident in the course of improvement, there is a corresponding decline in this postconvulsive agitation. Such an inverse ratio in behavior is, of course, not mathematically exact. But it appears to furnish an index of the degree of restraint and suppression which the sick person exercises over the impulses that he fears to express.

There are a number of observations occurring during electro-shock therapy which are interesting. Let us turn our attention to the memory impairment or amnesia. There is fragmentation of memory with com-

plete loss for recent events. The retention of memory follows an ontogenetic pattern of memory development:

1. Knowledge of personal name
2. Name of closest relatives: husband or father or mother or wife or children
3. Home address—but even here they may give a former home address instead of the present one, especially if the present one possesses some painful association, illustrating the mechanism of repression in amnesic states
4. Place of work or affiliation connected with work outside of home
5. The doctor's face will appear familiar but there may be prolonged difficulty in recalling his name. This is of peculiar interest since the patient will often remember the name of an assistant physician or nurse sooner than the physician administering the treatment. Here, again, we see the factor of repression in amnesia, to shut out painful impressions and associations
6. They recall the date, day, and month last, and with this recollection a flood of detail rushes back into conscious intellectual awareness. For example, they can then recall events which preceded the treatment during the morning or afternoon.

The order of memory retention is better understood when we point out that No. 2 and No. 6, for example, do not appear together immediately after the treatment. This would suggest that certain associational patterns in the areas of the cerebral cortex which subserve memory function are disturbed in the order of their temporal development. That is to say, the most recently developed memory for experiences is most vulnerable to disruption by electro-shock. This phenomenon, we recognize, is in accordance with Hughling Jackson's concept of dissolution of brain functioning occurring in the order of ontogenetic growth from primitive to more recently developed cerebral structures.

Although there are a number of points of psycho-physiological interest that lend themselves to extended discussion, mention will only be briefly made insofar as they are relevant to the subject of this paper.

First, is the general observation that each individual repeats a psychomotor pattern which tends to be characteristic for the individual. For example, one patient will manifest arm thrashing movements immediately after the cessation of the major fit in the first few treatments. Another will repeat leg thrashing movements, like a running pattern.

A number of patients, more likely those suffering from depression, will clench their jaws strenuously, resisting every attempt to remove the mouth gag. One patient used to reach out, in the twilight period of confused disorientation of semi-consciousness, and force the operator's hand to return the gag to his mouth. (The suggestive implications about oral drive are obvious). Many times one will observe sucking movements persisting after removal of the mouth gag, but I have never been able to elicit a genuine forced grasping reflex.

As they emerge into this twilight zone of semi-stupor from deep stupor, a pronounced *startle* pattern can be seen in a number of people when anyone approaches the treatment couch, or if someone speaks above a whisper. This resembles the wild primitive violent reflex of running amok, for the patient will shriek and shrink in terror or make belligerent gestures toward the approaching person. Another patient will begin to sing a bit of a child's song; this occurs seldom as most of the patients are mute and groan a bit and then seem to fall asleep. This sleep seems to be the most favorable type of after-convulsive reaction, for they are much more tranquil afterward, than if they show agitation and alarm about their disorientation during the semi-stupor.

Now, it may be very properly asked, "What is the meaning of this shock treatment for the patient?" And here we resort to material given by patients themselves. One said, "What have you done to me? I feel as though I'm reborn!" I have the impression that some patients react to shock treatment as a form of physical punishment, just as they may react to psychotherapeutic sessions as scoldings. Although the opinion has been expressed by some observers that patients do not seem to mind electro-shock treatment, I must disagree. While not comparable to the terror which metrazol held for patients, it has been my experience that electro-shock did frighten them. The level at which this dread was stored was unconscious usually, although some patients have told me that they consciously were afraid. They could not tell why, especially those who had major fits, but they sometimes would balk at taking a treatment. After a petit mal, particularly the brief and light variety, patients seemed very resentful and uncomfortable.

In my opinion, the shock treatment affects the psyche of the patient as a profound threat to his very existence. It reaches down to something primitive—we can call it the instinct for self preservation—or ego instincts—or what you will—in the human organism. Certainly, it is a

fact that they regard the loss of consciousness resulting from the treatment with dread; they feel that they must completely surrender themselves to the mercy of others as one undergoing general anesthesia. The same doubt as to whether they will emerge alive is operative in shock therapy. Significantly, a common reaction is to find early disappearance of suicidal impulses among the psycho-pathologic phenomena of the patient's mental disorder. One may speak of a reintegration of ego structure made possible by a violent shock to the personality.

With regard to the psychotherapy: should it be done by the same person who administers the shock? There are advantages and disadvantages in having the same doctor for both. One advantage is the matter of convenience; another is the opportunity to study every phase of the patient's behavior. It would seem of optimum benefit if the psychiatrist who is to administer psychotherapy could be present to assist the physician administering the electro-shock. In my experience, being the psychiatrist in addition to the one who also administers the electro-shock has not proven a serious obstacle in continued psychotherapy. Nevertheless, it is quite conceivable that a patient may be repressing resentment towards the electro-shock therapist to a degree which would make a desirable transference very difficult to attain for continued psychotherapy.

## Summary

There are instances in psychotherapy where the resistances of the patient are of such a character as to make an exclusively psychological approach unfeasible. In such cases, the supplementary value of electro-shock is to be borne in mind as a procedure which affects the rigidity of the psycho-pathological personality structure so that the patient is rendered more accessible to psychotherapy. The benefit derived from electro-shock seems to stem from psycho-physiological alterations which stimulate the individual's will to live and reduces the withdrawal tendency.

Finally, it may be said that whatever doubt may exist about the dynamics of improvement, there can be no doubt of the empirical fact that electro-shock therapy is an important contribution to the art and science of mental healing.

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# CERTAIN ABNORMALITIES OF OCULAR MOVEMENTS. THEIR IMPORTANCE IN GENERAL AND NEUROLOGIC DIAGNOSIS*

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DURING the past few years several clinical observations have added to our knowledge of ocular movements and ocular fixations. Since such observations have been described individually in different journals, redescription of them here may be permissible. Certain of them are important in regard to the cerebral integration of ocular movements—a subject which has been ably discussed by Holmes.[1] Part I of this paper is designed to review essential features which point to supranuclear lesions as the cause of abnormalities of conjugate movements and to redescribe relatively recent contributions in this field. The presentation is necessarily incomplete and concerns principles of diagnosis rather than precise anatomy. Part II contains a description of phenomena which arise through misdirection of regenerated fibers in the third nerve. These phenomena have been described previously by Bender,[2] Ford and Woodhall,[3] Bender and Fulton[4] and by Bielschowsky.[5] Their importance in the diagnosis of congenital aneurysms of the circle of Willis has been noted by Walsh and King.[6]

## PART I. (A) THE SUPRANUCLEAR MECHANISMS WHICH CONTROL OCULAR MOVEMENTS

The mechanisms which initiate and control conjugate movements of the eyes are described below under the following headings: (1) Frontal lobes and volitional control of conjugate ocular movements; (2) Occipital lobes and optic fixation reflexes; (3) Labyrinthine reflexes (a) semicircular canal reflexes, (b) otolith reflexes; (4) Tonic neck reflexes.

*Frontal Lobes and Volitional Control of Conjugate Ocular Movements.* Volitional movements of the eyes originate in cortical centers

* Read October 16, 1942 at the fifteenth Graduate Fortnight of The New York Academy of Medicine. From the Wilmer Institute of the Johns Hopkins Hospital, Baltimore, Maryland.

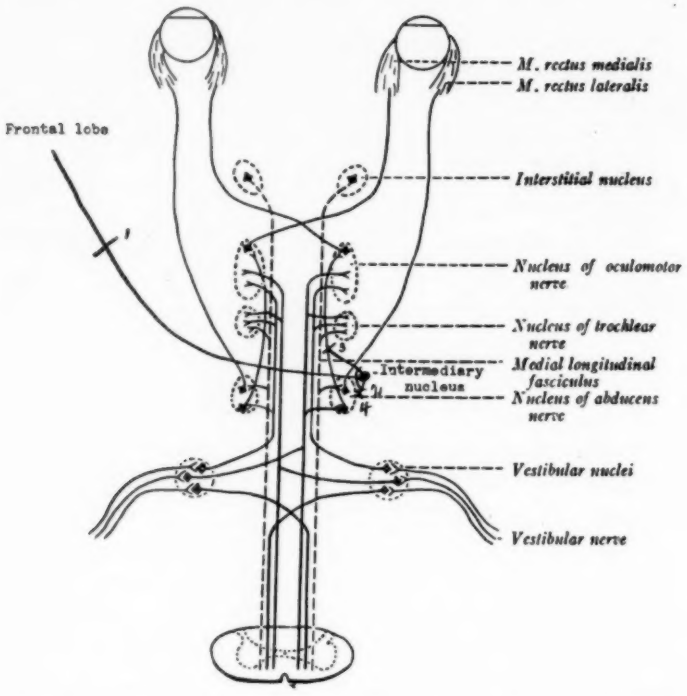



Fig. 1—Diagram showing the connections of the medial longitudinal fasciculus. (Modified from Villiger.) Further modified from Ranson to show results of lesions interfering with supranuclear pathway.

Lesion at 1 (in the hemisphere) results in conjugate deviation of the eyes to the side of the lesion.

Lesion at 2 results in conjugate deviation of the eyes to the side opposite the lesion.

Lesion at 3 results in anterior internuclear paralysis.

Lesion at 4 results in posterior internuclear paralysis.

termed "oculogyric centers" situated in the frontal lobe of each side, the area being frequently designated as area 8 (Brodmann). Electrical stimulation of this area on either side produces conjugate movement of the eyes (and usually the head) to the opposite side. If the lower part of the area of one side is stimulated there is upturning of the eyes in addition to the lateral movement, and if the upper portion is stimulated there is additional downturning added to the lateral movement. Ablations produce opposite results.

From these frontal cortical areas projection fibers enter the anterior

part of the internal capsule and reach the cerebral peduncles where they become detached from the corticospinal tract in small bundles at successive levels in the brain stem. They move dorsally to lie close to the medial lemniscus in the tegmentum. Then they undergo partial decussation and reach the intermediary supranuclear nucleus principally of the opposite side. It is from this level that impulses are mediated to the medial longitudinal fasciculus which is shown diagrammatically in Figure 1. Actually the position and course of the projection pathways from the frontal oculogyric centers has been postulated by Déjérine and never entirely confirmed. Further, it is fairly generally agreed that intermediary supranuclear nuclei exist but Collier[7] amongst others does not believe such nuclei are necessarily present.

*Occipital Lobes and Optic Fixation Reflexes.* When it was found that stimulation of the frontal lobe cortex produced contralateral conjugate movement of the eyes it was observed also that stimulation of the region surrounding the striate area likewise produced contralateral ocular movements. If, however, the frontal lobe and the opposite occipital lobe were stimulated at the same time the movement of the eyes always corresponded to the frontal lobe stimulation. Thus stimuli from the frontal lobes, the volitional centers, are stronger than stimuli from the occipital lobes. It is obvious that optic fixation centers must exist in the occipital lobes.

In the optic fixation reflex the afferent pathway is via the optic nerves and optic pathways. The corticofugal pathway is from the occipital cortex (mainly areas 18 and 19), down the internal sagittal stratum to the posterior limb of the internal capsule, round the inferior aspect of the optic thalamus to the superior colliculus and from thence to the brain stem. This pathway has been described by Kronfeld. It is remarked in passing that the optic fixation reflexes utilize cerebral cortex whereas vestibular reflexes apparently do not reach that level.

*Labyrinthine Reflexes.* The eyes may be conjugately deviated through the influence of sound as well as other sensations, but here we are concerned only with reflexes which are essentially vestibular in origin. These were analyzed by Breuer as long ago as 1875 with such exactitude that, as de Kleijn[8] has remarked, little has been added. Clinical observations are set forth below and they conform to the results of experiments performed by Breuer and confirmed by de Kleijn among others. Breuer divided vestibular reflexes into two principal groups

which are now described in brief.

(a) Semicircular canal reflexes are stato-kinetic, that is they produce a physiologic response to movement or to acceleration of movement. The stimuli probably arise through pressure of endolymph on the sensory end organs in the ampullae of the semicircular canals. Such reflexes account for brief contractions of skeletal muscles. In normal individuals this is exemplified in the occurrence of vestibular nystagmus which is essentially a jerky nystagmus. Semicircular canal reflexes have for long been known to be responsible for ocular fixations during movements of the head, but this knowledge has rested upon the results of experimental work until relatively recently. Since it has become known that section of the vestibular nerve or nerves is an effective method of treating Menière's disease evidence of pure vestibular dysfunction has been recognized clinically. Levin[9] described a unique case in which the vestibular nerves were selectively involved and in which the resultant symptoms were identical with those observed after surgical section of both vestibular nerves.

(b) Otolith reflexes are static reflexes and are responsible for tonic reactions. Magnus has shown that changes in the position of the head in its relation to space produces postural reactions which consist of assumed positions of the limbs and tonic deviations and rotations of the eyes. These reflexes arise in the labyrinth since they persist after the three upper cervical dorsal nerve roots on each side have been sectioned (de Kleijn), and they disappear after bilateral labyrinthectomy. Otolith reflexes seemingly arise as the result of weight of the otoliths and gravity. Consequently, turning the head into a lateral position does not call forth an otolith response because the position of the head is not altered in the field of gravity. When the head is flexed the eyes are deviated upward and when the head is extended the eyes are deviated downward as a result of otolith reflexes. When the head is inclined to one or other shoulder the eyes are rotated on the visual axis in the opposite direction to compensate for the altered position of the head. This last named evidence of otolith function is of real importance in the diagnoses of palsies of vertically acting muscles.

McNally and Tait[10] believe that the sacculus otoliths have nothing to do with tonic reflexes.

*Tonic Neck Reflexes.* These reflexes are of relatively slight significance in man, but they are most important in animals particularly those

in which the eyes are placed laterally in the head.

Tonic neck reflexes arise from proprioceptive nerve endings in the muscles of the neck as has been proven by their disappearance after section of the cervical nerve roots on each side. That tonic neck reflexes occur in the absence of labyrinthine reflexes is proven by their persistence after section of the vestibular nerves as was present in Levin's case, and as has been proven experimentally when they persist after destruction of the labyrinths. Tonic neck reflexes depend upon the position of the head in relation to the trunk. If the head is immobilized and the trunk is then moved, or if the labyrinths are destroyed and the head is moved, the tonic neck reflexes become apparent. In the latter instance, when the head is flexed, the eyes move upward and if the head is extended the eyes move downward, and if the head is inclined in one plane the eyes move in the opposite plane. From this it is apparent that tonic reflexes are supplementary to otolith reflexes and vice versa.

Labyrinthine and optic righting reflexes do not require consideration here.

## Part I. (b) Supranuclear Lesions and Their Effects Upon Ocular Movements

We are now in position to analyze the effects of certain supranuclear lesions from the standpoint of their influence upon ocular movements. Because of time limitation lesions influencing convergence and divergence are not considered.

It has already been remarked that experimenters abolish the function of one mechanism in order to study the function of another. Thus in order to study tonic neck reflexes it is necessary to abolish labyrinthine (otolith) reflexes and conversely if otolith reflexes are to be studied the tonic neck reflexes must be abolished. In the clinical study of supranuclear lesions, particularly when they are in the hemisphere, advantage is taken of the fact that suspension of volitional control enhances the optic fixation reflexes. If the eyes cannot be moved voluntarily into a desired position it is proven that the lesion is supranuclear if such movement can be obtained through the effect of some other mechanism which takes part in the movement but cannot be identified as participating in the normal individual. Since we are here concerned with abnormalities of ocular movements the subject of supranuclear lesions is considered from the standpoint of position of the eyes.

### I. LOSS OF VOLITIONAL CONTROL WITH CONJUGATE DEVIATION OF EYES TO THE SIDE OF THE LESION

Conjugate deviation of the eyes to the side of the lesion and inability to move them to the opposite side is present in a majority of cases of vascular accident when the lesion is situated in the hemisphere. To rationalize this statement it is only necessary to recall that electrical stimulation accounts for contralateral conjugate movements of the eyes, and that ablation accounts for conjugate deviation of the eyes to the side of the lesion.

With James I. Moore, I have studied more than fifty cases of cerebral hemorrhage in which there was a unilateral lesion. In such cases the lateral deviation of the eyes is pronounced. The deviated position is maintained in many instances for only a few hours and has always, in our experience, disappeared within a week or thereabouts. The head is usually deviated in the same direction as the eyes. In such patients, during the time that deviation of the eyes is present, there is loss of volitional control of the eye movements. Optic fixation reflexes are present and active when the patient is in contact, and in some cases the optic fixation reflexes are seemingly exaggerated. Thus they may be utilized in moving the eyes into positions otherwise unobtainable, and such movement is proof of the supranuclear origin of the lesion. Bilateral hemispheric lesions may account for complete inability to move the eyes conjugately or separately in any direction through voluntary effort. In such cases the defect is usually more or less permanent.

Various maneuvers are undertaken to obtain movement of the eyes into otherwise unobtainable positions insofar as the patient's efforts are concerned. Since in actual practice these maneuvers are undertaken to bring either optic fixation reflexes or labyrinthine reflexes into active functioning the various procedures are now outlined.

If a patient exhibits pronounced deviation of the eyes and head to the right there is inability to voluntarily move the eyes to the left. The following maneuvers may suffice to obtain the voluntarily impossible movement of the eyes.

1. The patient is asked to fix the light on a test object which is moved slowly in front of the eyes from right to left. In some instances the eyes will follow the light. This is called the following movement.

2. With the patient fixing the light, the head is grasped in the

examiner's hand and is slowly rotated in the appropriate direction. The eyes may move into the desired field.

If volitional control of the eye movements is not entirely lost but is weakened the resulting relative efficiency of the optic fixation reflexes may account for inability to move the eyes until blinking interrupts the fixation reflex.

3. In some instances where voluntary movement of the eyes is impossible, if a light is held in the seemingly unobtainable field, the eyes are immediately turned toward the light. Bielschowsky[5] considered this to be an optic fixation reflex.

From consideration of the tests mentioned above it becomes apparent that loss of volitional control in some instances enhances the optic fixation reflexes and may make them relatively excessive. However, it is not always possible to use the optic fixation reflexes since the patient may be unconscious; he may refuse to coöperate although conscious, or the optic fixation reflexes may not serve to obtain the desired deviation of the eyes. In such instances other maneuvers may be employed and in these the vestibular reflexes are utilized.

4. The patient's head is grasped by the examiner and is rapidly rotated when it may be noticed that the eyes deviate momentarily into the position desired by the examiner. This reflex movement is thought to be due to reflexes arising in the semicircular canals for it is of brief duration and is entirely different to that elicited by slow rotation of the head with the eyes fixing as has been mentioned above (2).

5. The caloric test may be used. If, in the example being considered, namely when the eyes are deviated to the right, the left ear is syringed with cold water the eyes move conjugately to the left. This is providing the paralysis of ocular movement is supranuclear in origin and originates from a lesion in the hemisphere, and further providing the vestibular pathways are intact.

The caloric test has certain advantages over rotation tests which are not considered in this paper. One of the most obvious advantages is that the caloric test can be performed on patients who cannot be placed in a rotating chair.

### II. LOSS OF OPTIC FIXATION REFLEXES WITH RETENTION OF VOLITIONAL CONTROL

Lesions which interfere with functioning of the posterior oculogyric

apparatus may abolish the capacity for optic fixation without interfering with visual acuity or with the visual fields. Since, however, such lesions are likely to interfere with the striate cortex and (or) with the optic radiations, loss of optic fixation reflexes is rarely observed as an isolated symptom. Abnormal functioning of the posterior oculogyric apparatus is seen much less often than involvement of the anterior oculogyric apparatus.

The symptomatology of lesions affecting the posterior oculogyric apparatus has been well illustrated in a case described by Holmes and Horrax. Their patient, a soldier suffering from the effects of a wound involving the optic pathways, exhibited loss of a quadrant of the lower visual field in each eye. Central visual acuity was normal and there was no evidence of paralysis of the extraocular muscles. The following features were described: (1) There was difficulty in fixation. He could maintain fixation on an object when he found it but there was great difficulty in locating it. (2) Accommodation-convergence was affected. If an object were brought close to his eyes he could not converge his eyes on it, but if the object were his own finger convergence was performed normally. (3) When objects were shoved close to his eyes he did not blink unless the object happened to be his own hand when blinking was prompt. (4) Visual word memory was altered. He was unable to describe the manner in which he found his way about in trenches with which he was familiar. (5) While he could recognize movements of objects he did not know whether they were being moved to or from him. (6) He could read only with difficulty and could not find the start of the following line. (7) Stereoscopic vision was lost. (8) Volitional movements of the eyes were not influenced. With Frank R. Ford, I have described a somewhat similar case in which there was loss of the optic fixation reflexes as a result of nitrous oxide anesthesia.

At this time a generalization may be made regarding the differentiation of lesions involving the anterior and posterior oculogyric mechanisms. Lesions interrupting the anterior apparatus produce relative increase in the optic fixation reflexes and conjugate deviations to the side of the lesion when it is destructive and in the hemisphere, and to the opposite side when it is irritative. Lesions involving the posterior oculogyric apparatus, often complicated by lowering of visual acuity and field defects, result in inability to properly fixate objects with the eyes but not in interference with volitional movements of the eyes.

### III. LOSS OF VOLITIONAL CONTROL WITH DEVIATION OF THE EYES AWAY FROM THE SIDE OF THE LESION

As has already been described when the lesion is in the hemisphere the eyes are directed toward the side of the lesion and there may be inability to move them to the opposite side. Since, however, the projection fibers from the frontal lobe cortex cross in the midbrain for the most part, a lesion in the midbrain accounts for deviation of the eyes away from the side of the lesion and for paralysis of volitional movement of the eyes to the side of the lesion. There are other important differences between lesions at this level and hemispheric lesions and I cannot do better for description than include Bielschowsky's table.[5]

LATERAL CONJUGATE DEVIATIONS OF THE EYES (BIELSCHOWSKY)

| *Lesions in the Hemisphere* | *Pontine Lesions* |
|---|---|
| 1. In the first stage regular and of considerable magnitude. | 1. Relatively rare and as a rule of small magnitude. |
| 2. Deviation usually of short duration. | 2. Deviation, if present, permanent. |
| 3. Deviation toward the side of the lesion. | 3. Deviation toward the opposite side. |
| 4. Deviation frequently a sign of stimulation. | 4. Deviation usually a paralytic symptom, only rarely a sign of stimulation. |
| 5. Head turned (as a regular symptom) in the same direction as the deviation of the eyes. | 5. Abnormal position of the head not typical symptom if present. |
| 6. Associated paralysis of muscles for contralateral movements, usually slight and transient. | 6. Associated paralysis in the direction of the lesion always severe and permanent. |
| 7. Invariably symmetrical functional disturbances of associated muscles. | 7. Frequently asymmetrical paralysis of associated muscles in consequence of extension of the supranuclear lesions to nucleus or nuclei. |
| 8. Paralysis of extremities and of facial nerve collateral with the associated eye muscle paralysis. | 8. Paralysis of extremities, if present, opposite to the side of the eye muscle paralysis. Paralysis of the facial nerve, if present, usually collateral with the eye muscle paralysis. |
| 9. In lesions of both hemispheres all eye movements (including the vertical) restricted or impossible. | 9. In pontine lesions of both sides paralysis of side to side movement without paralysis of vertical movements. |

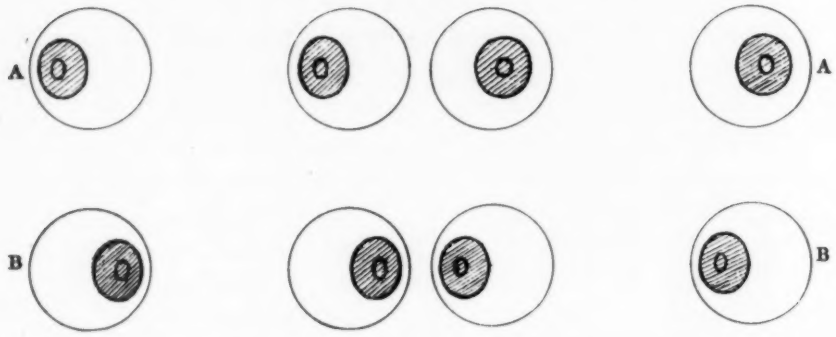



Fig. 2 (1)—A. Conjugate deviation to the side of the lesion: lesion in hemisphere: paralysis volitional movement to left. B. Eyes may be moved to left: (1) by utilizing optic fixation reflexes; (2) by impulses from semicircular canals (quick rotation of head); (3) by caloric stimulation (cold water in left ear); (4) by proper rotation.

Fig. 2 (2)—A. Conjugate deviation away from side of lesion (lesion in pons): paralysis volitional movement to the right: convergence ability retained. B. Eyes may be moved to right by caloric stimulation right ear and by proper rotation tests.

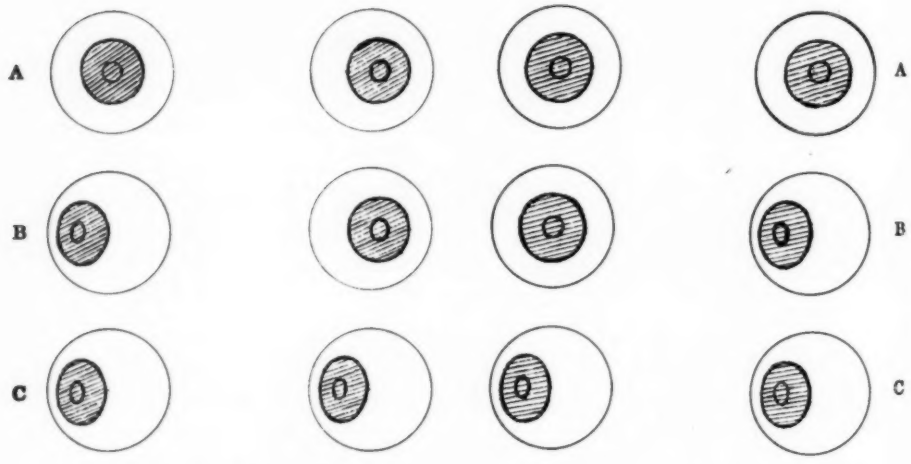



Fig. 2 (3)—Anterior internuclear paralysis. A. Eyes straight: convergence ability retained; B. Eyes right: left eye fails to turn to right; C. Left eye turns to right after caloric stimulation (cold water right ear).

Fig. 2 (4)—Posterior internuclear paralysis. A. Eyes straight; B. Eyes right: left eye turns to right but right eye fails to do so; C. Right eye turns to right after caloric stimulation or rotation.

Fig. 2. Diagrams illustrating paralyses of lateral conjugate movements (lesions on right).

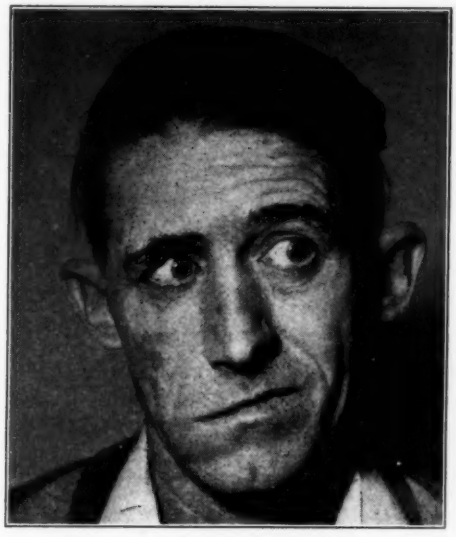


Fig. 3—T. Z. Patient suffered from glioma of the pons. There was paralysis of the right 6th, 7th and 8th nerves, paralysis of conjugate movement to the right. The eyes could be converged but caloric stimulation of the right vestibule failed to produce adduction of the left eye. Doubtless the vestibular tract on the right had been destroyed.

We may now consider abnormalities of lateral conjugate movements of the eyes as the result of lesions in the brain stem. If there is a unilateral supranuclear lesion which affects conjugate ocular movements it may involve: (1) the supranuclear intermediary center in the region of the sixth nerve nucleus. If the lesion is on the right there is slight deviation of the eyes to the left and they cannot through volition be moved to the right. The power of convergence is retained. If the right ear is syringed with cold water the eyes usually move freely to the right. (2) The lesion may involve not only the supranuclear center of one side but may invade the sixth nerve nucleus and nuclei of other cranial nerves usually on the same side. Figure 3 shows such a lesion. The patient suffered from a glioma of the pons. There was paralysis of the right sixth, seventh and eighth nerves. The eyes could be converged. Caloric stimulation (cold water into the right ear) failed to produce adduction of the left eye hence the vestibulo-ocular pathway was also

interrupted. (3) The lesion may be situated in such a position that it interferes with impulses passing to the opposite internal rectus muscle. Such a lesion usually is in the medial longitudinal fasciculus. This comprises an anterior internuclear paralysis. (4) The lesion may be situated in a position to interfere with impulses reaching the sixth nerve nucleus while not interrupting impulses reaching the contralateral internal rectus muscle. This comprises a posterior internuclear paralysis. (5) There may be bilateral lesions which involve both supranuclear centers in the pons. In this instance there is inability to move the eyes voluntarily either to the right or left, but vertical movements and the ability to converge are retained. In such instances caloric stimulation serves usually to deviate the eyes in the desired direction. I have observed such a bilateral paralysis of supranuclear type in a child suffering from glioma of the pons. Bilateral anterior internuclear paralysis has been described by Spiller. His patient could not adduct either eye during attempts at lateral conjugate movements but was able to converge the eyes normally. Probably bilateral posterior internuclear paralysis has been described but if so I have overlooked the description.

It has been said that as a result of hemianopsia there may be conjugate deviation of the eyes. Although deviation of the head undoubtedly is observed frequently in association with hemianopsia in order to increase the size of the visual field I have not observed conjugate deviation of the eyes in the absence of deviation of the head in such cases. The patient with hemianopsia may exhibit awkwardness in looking toward the blind field but this should not usually be confused with paralysis or paresis of conjugate movement.

### IV. LOSS OF ABILITY TO VOLUNTARILY ELEVATE AND DEPRESS THE EYES

Up to this point only abnormalities of lateral conjugate movements of the eyes have been mentioned. Of equal importance, but of less frequent occurrence in my experience, is inability to voluntarily elevate and depress the eyes. Such ocular paralyses arise as the result of supranuclear lesions.

The mechanisms through which volitional upward and downward movements of the eyes are obtained are not so readily visualized as those for lateral movements. Holmes[11] has predicated the existence of intermediary centers in the midbrain at about the level of the superior colliculus. That for upward movements, he states, is anterior, and pro-

gressing downward there is a center for downward movement and one for convergence.

Inability to voluntarily direct the eyes upward or downward is commonly described as Parinaud's syndrome. The diagnosis of supranuclear lesions for such a disability requires mention of further maneuvers or tests not as yet mentioned.

6. With the eyes fixing, the patient's head is slowly flexed when the eyes may turn upward, or if there is inability to look downward extension of the head may account for downturning of the eyes.

7. Bell's phenomenon may be exhibited as proof of the supranuclear origin of the paralysis. The patient is asked to close the eyelids while the lids are held open by the examiner. If Bell's phenomenon is present the eyes are turned upward.

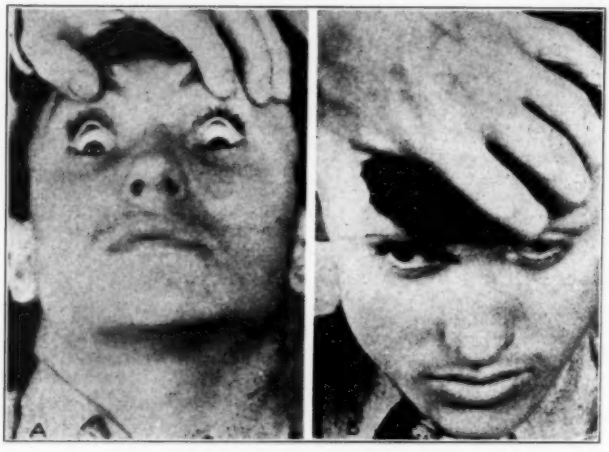
8. If caloric stimulation is applied while the patient holds the head flexed as far as possible, or preferably in our experience if the head is tilted to one or the other shoulder, tonic upward deviation of the eyes may be obtained. A similar result is obtained when rotation is performed.

The presence of Parinaud's syndrome suggests a lesion in the region of the quadrigeminal plate, often a pineal tumor. We have observed the development of a typical case in a young girl. The first abnormality noted was wide dilatation of the pupils which failed to react to light. Then there was inability to voluntarily look upward, and finally there was inability to look downward as well. Parinaud's syndrome may be observed in association with ptosis and small actively reacting pupils as we have observed. In some instances the syndrome is transient as has been observed by Dr. Arnold Knapp and by the present writer. In such cases there may be an associated unilateral or bilateral proptosis in rare instances.

While on the subject of paralysis of upward and downward movement of the eyes as caused by supranuclear lesions mention may be made of a unique case which has been described by Dr. Ford and the writer.[12] The clinical studies on this case followed the pattern of studies made by experimenters on animals. This case appears to stand alone as clinical proof of the function of the otolith organs in man.

Case 1. *Congenital double athetosis associated with congenital paralysis of upward and downward movements of the eyes in a boy 13 years of age; elevation and depression of eyes obtained by active and passive movements of the head.*

Case 1—Volitional movements of the eyes. *A* shows full range of movement to the right, and *B*, full range of movement to the left.



Case 1—Deviations due to movements of the head. *A* shows deviation of the eyes downward when the head is passively extended, and *B*, deviation upward when the head is flexed.

Case 1—Reactions on the tilting table. *A* shows upward deviation of the eyes due to bringing the patient from a horizontal to an upright position. *B* shows downward deviation of the eyes due to rotating the patient from an upright position backward to a horizontal position.

Fig. 4—(Case 1). Congenital double athetosis associated with congenital paralysis of upward and downward movements of the eyes in a boy 13 years of age; elevation and depression of eyes in the orbits obtained by active and passive movements of the head. (Ford and Walsh.)

L. S. was examined on June 15, 1939. Neurologic examination revealed the usual signs of double athetosis which was known to be stationary and had been noted in early childhood. It had never been possible for the child to make satisfactory progress in learning to read despite the fact that his intelligence seemed to be normal. His teachers stated that the difficulty seemed to lie in his inability to move the eyes properly. Examination of the eyes yielded the following observations:

1. Vision and visual fields were normal. The pupillary reactions were brisk. The optic fundi were normal.

2. Voluntary movements of the eyes to the right and left were normal. He could follow a moving object to either side.

3. Voluntary movements of the eyes downward below the horizontal were absent. When fixation was prevented by closing the eyes there were still no downward movements.

4. Voluntary movement of the eyes upward was difficult and was limited to less than 10 degrees.

5. Convergence was entirely absent.

6. It was impossible for the patient to follow a test object moving slowly in the vertical plane. This test was made repeatedly. The revolving drum failed to elicit any reaction when it was revolved about a horizontal axis.

7. When the patient's head was extended actively or passively, compensatory deviation of the eyes downward occurred. By this means the eyes might be deviated to the limit of their normal excursion. In the same manner flexion of the head caused upward deviation of the eyes. These reactions were secured just as easily when the eyes were closed as when they were open and by slow movements of the head as well as by rapid movements. They were constant and never varied in the slightest degree.

8. The patient was placed on a tilting table in such a manner that no movement of the neck could occur. When he was rotated from an upright position backward the eyes deviated downward just as when his head was extended on his body. When he was rotated from a horizontal to a vertical position his eyes deviated upward just as when his head was flexed on his body. It was clear that it was the change in the position of the head in space and not any movement of the head on the body that caused the deviation of the eyes. The ocular deviations were well sustained.

9. The patient had learned an interesting trick by which he could deviate his eyes downward. When asked to look at an object in his lap he would flex his head and the upper part of his trunk as strongly as possible. During this movement his eyes were, of course, rotated upward in the orbits, but by extreme flexion of the spine he could finally bring them to bear on the test object, for the range of movement of his head was greater than that of his eyes. At this point he would bring his head into the upright position and his eyes would remain fixed on the test object in the position of downward deviation. The examiner could reproduce this reaction by making passive movements of the patient's head. It was also possible to fix the patient's eyes on a test object above his head by making extreme extension of the head and then bringing it into an upright position. The patient failed to discover the latter maneuver until it was shown him.

10. Bell's phenomenon was present.

In the case described above it seemed that otolith reflexes were responsible for the tonic deviations of the eyes for the following reasons: (1) There was loss of optic fixation reflexes in the vertical plane as demonstrated by absence of optikokinetic nystagmus with the axis of the drum placed horizontally. Furthermore optic fixation reflexes could not have produced the tonic deviations because the position of the eyes changed when fixation was abolished by closure of the lids. (2) The possibility of semicircular canal reflexes being responsible was eliminated by the tonic nature of the ocular deviations. Semicircular canal reflexes are of brief duration. (3) Tonic neck reflexes were ruled out by observation of upward and downward deviation of the eyes when the patient was placed on the tilting table. (4) The reactions were in the field of action of the otolith organs, that is they were produced by alterations of the position of the head in space, and were sustained.

The second case which is described in summary illustrates the result of section of both vestibular nerves. It is to my knowledge the first such case to have been described (Ford and Walsh[13]).

Case 2. *Man 43 years old developed paroxysms of vertigo with mild reduction of hearing and some tinnitus. Operative section of the vestibular components of both eighth nerves with complete relief of original symptoms. Postoperative development of unsteadiness on turning head and uncertainty when crossing streets. Loss of ocular fixation dur-*

**Fig. 5—Regeneration with misdirection of fibers. When adduction or downward movement was attempted the lid became elevated. Case of saccular aneurysm of right internal carotid artery, in a man aged 58, treated by partial ligation of artery in neck and a month later by intracranial ligation; degeneration of third nerve producing abnormal movements of lid associated with ocular movements; partial recovery from internal ophthalmoplegia; abnormal pupillary movements. (Walsh and King[4])**

*ing movements of the head. No change in hearing, but complete loss of all vestibular reflexes.*

J. B., 43 years old, developed paroxysms of vertigo often accompanied by vomiting which sometimes lasted a day or more. There was slight tinnitus aurium on either side and mild reduction of hearing in both ears, more pronounced on the left. The vestibular reflexes were normal. Neurological and medical examination revealed nothing of importance except for mild hypertension. A diagnosis of paroxysmal aural vertigo or Meniérè's disease was made. The patient was treated

by various conservative methods without result.

Craniotomy was performed and the vestibular components of both eighth nerves were severed. This put a stop to the paroxysms of vertigo and resulted in complete loss of all vestibular reactions on turning and irrigations of the ears. The audiometer chart showed no change in hearing following the operation.

After operation it became apparent that a new series of symptoms had developed. Objects seemed to move before his eyes unless his head was kept perfectly still. Walking caused objects to "jump" before his eyes to some extent, but this did not interfere with his gait. He was unsteady because he had to turn his head to look for approaching cars. Consequently he had become accustomed to hold his head very stiffly and walk in straight lines looking directly before him. He was unsteady in the dark. These symptoms were unchanged after 14 months. Vestibular stimulation either in the form of caloric stimulation or rotations failed to produce nystagmus. The optic fixation reflexes were responsible for normal optikokinetic nystagmus.

*Summary Part I:* The abnormalities of conjugate ocular movements and of optic fixations have been described from the standpoint of supranuclear lesions situated at different levels. The function of the semicircular canals in controlling optic fixations during movements of the head has been described from a clinical standpoint, as has a case illustrating the normal function of the otolith. Emphasis has been given to the few procedures which point to abnormalities of ocular movements being caused by supranuclear lesions.

## Part II. Misdirection of Regenerated Fibers in the Third Nerve

Misdirection of fibers during regeneration results in some instances in anomalous movements of the upper lid, abnormalities of movements of the globe, and in pupillary anomalies. These phenomena may properly be attributed to misdirection as has been described by Bielschowsky,[5] Bender,[2] Bender and Fulton[4] and Ford and Woodhall.[3] Walsh and King[6] remarked upon the importance of the misdirection phenomena in the diagnosis of congenital aneurysms of the circle of Willis.

During regeneration, as has been pointed out by Cajal and others, there is multiplication of nerve fibers. These fibers become misdirected or mixed in their distribution. Thus fibers designed to reach the internal rectus muscle may reach the superior rectus while others intended for

the inferior rectus may reach the levator palpebrae and so on. This hypothesis is easier to prove in the instance of regeneration in the third nerve than it is in the seventh where it accounts for mass facial movements.

The following observations may be made regarding the lids and ocular movements in a case of misdirection after recovery from third nerve paralysis. (1) The patient can adduct the eye normally. The internal rectus muscle is not opposed by any muscle innervated by the third nerve, hence the normal internal movement. (2) The eyeball cannot be elevated or lowered because the superior and inferior rectus muscles oppose each other actively. That there is not a paralysis of either of these muscles has been shown experimentally by Bender and Fulton.[4] If they sectioned the superior rectus muscle the eye could be lowered. (3) When the eye is adducted the upper lid is elevated. The levator palpebrae is unopposed by any muscle innervated by the third nerve hence the outflow of impulses causes it to elevate sharply when the eye is adducted either in convergence or in lateral movement. (4) The upper lid is lowered when the eye is abducted. This is in conformity with Sherrington's dictum regarding reciprocal innervation of the extraocular muscles.

The following observations have been made by Ford, Walsh and King[14] regarding pupillary movements after third nerve regeneration. (1) The pupil may be large and fail to react to light on convergence. (2) The pupil may be either larger (usual) or smaller (rare) than its fellow and may fail to react on direct light stimulation but reacts during convergence and any movement during which the eye is adducted. (3) The pupil may react sluggishly to light and more actively during convergence or any movement in which the eye is adducted. (4) The pupil in the affected eye often dilates during abduction of that eye. This again exemplifies the validity of Sherrington's observations regarding reciprocal innervation.

In commenting upon the pupillary phenomena which are associated with regeneration of the third nerve it has been remarked that one of the pupillary anomalies resembles the Argyll Robertson pupil. Furthermore, it is of particular interest that regenerating somatic motor nerve fibers may establish effective synapses with post-ganglionic neurons of the autonomic system.

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# THE MANAGEMENT OF THE PATIENT WHO HAS RECOVERED FROM ACUTE CORONARY OCCLUSION*

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CLOSURE of a coronary artery or one of its branches may result from various causes. The most common pathological processes responsible are atherosclerosis and thrombosis.[1] This discussion will be confined to a consideration of acute obstruction followed by infarction of the myocardium due to such lesions. The anatomical changes which subsequently take place in the heart muscle are directly related to the course of the illness and, consequently, to therapeutic procedure. Reference will be made to them in appropriate places.

When may it properly be said that a patient has recovered from the acute phase of an attack? The uncertain period following cardiac infarction is during the first two weeks, for it is then that serious and unforeseen upsets are most likely to occur. Among these are sudden death due to ventricular fibrillation or to abrupt cardiac standstill; rupture of the heart wall; various arrhythmias, particularly ventricular tachycardia and auriculoventricular heart block; and embolization to the lungs or to a peripheral artery, of which the source is a mural thrombus within one of the ventricular cavities. If the third week has been completed without mishap, the members of the patient's family may breathe more deeply and sleep more soundly, since they can be assured that the outlook for recovery is favorable. But the depth of the physician's sigh of relief, even at this time, is modified by his estimate of the severity of the damage to the heart. Familiarity with the amount of cardiac and coronary reserve which was present prior to the acute episode is of little help in making this estimate; clinical symptoms and signs, and the trend following the attack are the only dependable guides.

* From the Department of Medicine, College of Physicians and Surgeons, Columbia University, and the Medical Clinic of the Presbyterian Hospital, New York. Lecture given as part of a Refresher Course in Cardiovascular Diseases, under the joint auspices of The New York Academy of Medicine and the New York Heart Association, November 25, 1942.

Shortly after occlusion has occurred it is impossible, even for the experienced observer, to make an accurate prognosis with respect to survival or the degree of subsequent functional recovery. As the days lengthen into weeks and the course can be charted, the details of the picture are gradually filled in. Usually, by the end of the second or third week, the case may be fitted into one of four main clinical categories; management of the stage of healing and the later period of convalescence will depend, in large measure, upon such classification. Viewed in this way the broad principles of treatment, rather than its minutiae, form the basis of procedure. The types of reaction are not sharply defined and some of their characteristics overlap; but they serve usefully in a practical sense and may be described briefly as follows:

*Group 1. Critical, with shock.* The outcome is always in doubt. Serious complications often occur. The heart is permanently crippled. Death, as a rule, is caused eventually by sequelae of the cardiac injury.

*Group 2. Severe, with shock.* The patient is gravely ill for several weeks. Convalescence is prolonged but there is partial, and often fairly good, functional recovery. It is unusual, however, for life to be prolonged for more than five years.

*Group 3. Moderate, without shock.* Although the attack may be stormy, the signs are favorable before the end of the second week. Functional recovery is good, sometimes complete, and there is frequently a long period of survival.

*Group 4. Mild, often unrecognized.* Some of these patients are ambulatory throughout. Discomfort at the time of the attack is not great or prolonged. Functional recovery is usually complete; but if the accident is ignored, life may terminate suddenly and unexpectedly before healing has taken place.

To formulate rules of action applicable to every contingency is manifestly impossible. Instead of attempting to present a formal catalogue of suggestions, it has seemed more profitable to cite the histories of cases illustrating each of the four categories and to discuss certain details of management relating to them.

### The Critical Type, With Shock

*Case 1.* A trained nurse, aged forty-three years, claimed never to have known a sick day before the onset of the present illness. She had been a heavy smoker. While moving furniture in her apartment,

she felt a sudden, crushing, substernal pain, radiating down both arms, and this was followed by profuse perspiration. She vomited and thought she would die. A physician gave her large doses of morphine. Vomiting continued at intervals for thirty-six hours. An electrocardiogram was typical of early anterior infarction. She remained at home in bed during the next three weeks and while there, had repeated episodes of substernal pressure and shortness of breath. On the twentieth day, because there was no improvement, she was moved to the hospital.

On admission, she was still having pain and dyspnea, with a respiratory rate ranging from thirty to forty per minute. The rectal temperature was 102° F., the leukocytes were increased and the sedimentation rate was prolonged. There was slight cyanosis and numerous rales were heard at the bases of both lungs. The blood pressure was 130/90. Her condition was regarded as precarious and she was immediately placed in an oxygen tent.

During the following months there were repeated paroxysms of dyspnea and numerous hemoptyses, associated with pain in the chest. Dullness developed over both lower lobes and there were signs of congestive heart failure. The blood pressure fell to 106/52 and the venous pressure rose. Although there were fluctuations in the general condition, the trend was downward. The leukocyte count, which at the end of a month had returned to normal, again rose and remained in the neighborhood of 14,000, with 75 to 80 per cent polymorphonuclears. Fever continued, though it was not high. She was kept in the oxygen tent for three weeks. Digitalis was given in full doses and a daily maintenance ration was continued. Sedatives, in the form of bromide and luminal, were necessary during the daytime and were supplemented by hypodermic injections of morphine at night, and whenever discomfort became extreme. On several occasions, the increasing edema was lessened by intravenous injection of mercupurin, followed by theocalcin given by mouth, but improvement was only temporary. She became emaciated and weaker, and finally died of cardiac insufficiency eight months after the onset of illness.

The necropsy revealed adequate cause for the progressive failure. The heart weighed 430 grams. The anterior descending branch of the left coronary artery contained a large, yellow intimal plaque, which reduced its lumen by about one-half. The remainder of the

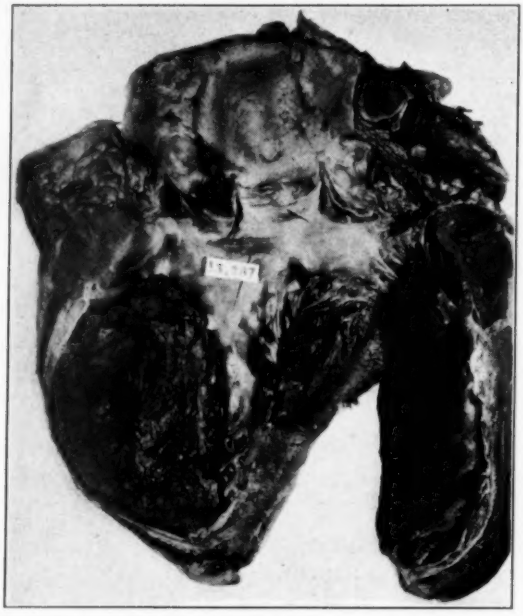

Fig. 1. Photograph of the heart of Case 1, showing the thinned, bulging wall of the left ventricle and the large intraventricular thrombus.

lumen was occupied by an organized thrombus. There was a large area of infarction involving the anterior part of the lateral wall of the left ventricle, as well as the interventricular septum. The infarct was healed but the wall of the left ventricle was thinned and bulging (Fig. 1). The other large branches of the coronary arteries showed advanced sclerosis, with numerous intimal plaques, some of which were calcified. The lumina were considerably reduced, but nowhere else was there complete obliteration.

Of striking interest was an enormous thrombotic mass occupying about half of the left ventricular cavity and situated in its anterolateral portion. It was firmly attached to the underlying endocardium. Almost half of the lower lobe of the right lung was solid, due to numerous infarcts, and there were scattered areas of infarction also in the lower lobe of the left lung. Arteriosclerotic scars were present in both kidneys.

*Discussion.* In this case, no therapy was effective. A large portion of the wall of the left ventricle was damaged and the function of the heart

was further impaired by the intraventricular clot. In addition to anoxemia caused by cardiac failure, there was deficient oxygenation of the blood as a result of extensive infarction of both lungs. The administration of oxygen in high concentration for several weeks,[2, 3] and attempts to combat congestive failure with the use of digitalis, mercupurin and theocalcin, failed to bring about lasting improvement, although life was prolonged for eight months. The chief aim of therapy was to maintain the patient in a state of relative comfort. The extent and nature of the pathological lesions determined the inevitably fatal outcome.

### The Severe Type, With Shock

*Case 2.* A business executive, aged sixty-one years, had been unusually active. For several months prior to the onset of the present illness, he had experienced moderate precordial pain on exertion, but kept at work. His attack of coronary occlusion was sudden and was followed by the classical symptoms of shock. He was placed immediately in an oxygen tent, where he remained for two weeks. The concentration was maintained between 50 and 60 per cent. There was a marked fall in blood pressure and the heart sounds were feeble. For a week there was doubt as to whether he would survive. Edema of the ankles and sacral region developed and he was, accordingly, given full doses of digitalis, namely one cat unit three times a day for one week. This was followed by disappearance of the edema, improvement in the character of the heart sounds and some elevation of blood pressure. He was kept flat on his back for four weeks and was then gradually permitted to sit up in bed for short periods, but did not get out of bed until the end of the eighth week. He developed severe pain in the right shoulder, which was partially relieved by the use of the infra-red lamp for twenty minutes twice a day, and by massage. He was first allowed to sit in a chair for fifteen minutes a day and then for gradually increasing periods. During the subsequent weeks, edema recurred and was treated successfully by digitalis and intravenous injections of mercupurin. A maintenance dose of digitalis was continued. At the end of four months, he went to Florida where he remained for twelve weeks. While in this warm climate, he walked for short distances and was permitted to take dips in a heated pool, never going above his depth. He was given light general massage three times a week. He was not permitted to begin work for six months after the onset of his illness.

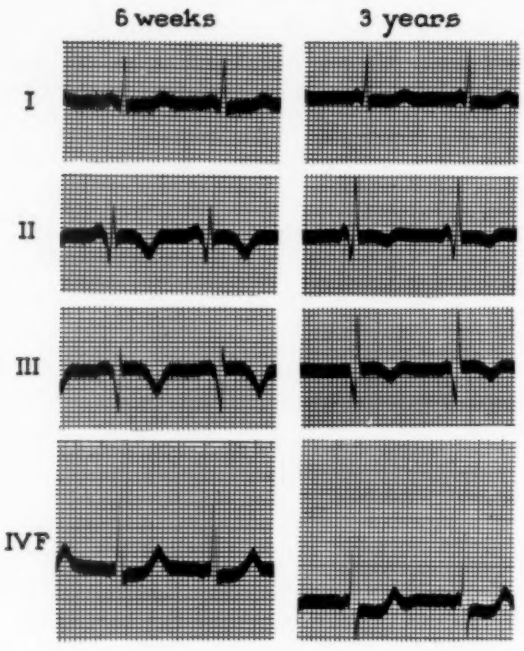


Fig. 2. Electrocardiagrams of Case 2 after the acute attack, taken at the intervals indicated. The second record was made two days before death.

There was no return of heart failure. The administration of digitalis was stopped. The blood pressure rose to normal levels. He became more active in business; when he was tired, premature beats of ventricular origin were noted. These were readily controlled by the use of quinidine sulphate, 3 grains taken three times a day after meals.

During the next three years, the patient took long winter and summer holidays. There was never any pain, although at times he was aware of a sense of pressure in the substernal region, both after effort and when he was under emotional stress.

Three years after his attack, while attending the wedding of a relative in another city, he partook freely of food and drink. That night he had an acute upset with digestive symptoms, and died suddenly a few hours later, undoubtedly as the result of another attack of coronary occlusion.

The electrocardiogram of this patient shows, in the first tracing

pictured, changes due to posterior infarction. The second record, taken three years later, shows these changes to be less in degree, but still present (Fig. 2). It was two days after this record was made that he died.

*Discussion.* The length of time that a patient should remain in bed after he has suffered an attack of cardiac infarction is variable. It is now the usual practice to make it six weeks; but this time may be and should be varied according to the circumstances in each case. What are some of the guides which help in reaching a decision? When there have been symptoms of shock at the onset, this period should not be shortened. When there have been signs of congestive failure it should be prolonged to a point at which all such signs have disappeared. There is pathological evidence that small infarcts heal almost completely in a month; larger infarcts require up to two months and there are instances in which areas of necrotic muscle may remain longer as foreign bodies in the midst of myocardial scars.[4] The leukocyte count rises promptly following occlusion and is increased as the area of necrosis grows larger. On the average, and in the absence of complications, leukocytosis disappears in the course of five days. One of the best guides to the rate of healing of an infarct is the sedimentation rate of the erythrocytes.[5] This usually does not rise significantly until the third or fourth day, reaching its peak between the fourth and eighth days. It reflects delicately both healing and any extension of the process. In the average case, it does not return to normal for about a month. When it remains elevated longer than this, it is well to suspect either incomplete healing, the presence of an intracardiac thrombus (as was described in Case 1), or the occurrence of some complication, such as pulmonary infarction. As a general rule, it is wise to keep the patient in bed until the sedimentation rate has reached a normal level. The effect of a known factor which might modify the reading, as for example, cystitis, must be discounted.

The rate of the heart is another useful guide. Usually this returns to normal in the course of ten days. Persistent tachycardia, particularly if associated with a diastolic gallop rhythm, is an index of severe cardiac damage and calls for more than the usual period of bed rest.

When the patient first gets out of bed, it is helpful, in order to prevent the collection of blood in extremities grown flabby from disuse, to apply Ace bandages to the feet and legs. These may be put on

before the patient leaves his bed and removed after he has returned to it; continue bandages during the first week that the patient is up.

A painful shoulder following coronary occlusion is not uncommon.[6] It is not due to a local lesion, but is probably dependent upon afferent impulses from the heart which stimulate neurones whose fibers form part of the brachial plexus. There is tenderness and limitation of motion, but usually there are no changes in the x-ray picture, either in the joint or in the supraspinatus tendon. The pain may persist for weeks and sometimes for months. It sometimes causes discomfort at night, when the patient is awakened by rolling over on the tender area. The left shoulder is more commonly affected than the right, but the right is not immune. Heat, in the form of diathermy or the infra-red lamp, massage and passive movement afford some relief. There is a tendency for discomfort gradually to abate.

Guidance during late convalescence and during the years that follow requires understanding and patience on the part of the physician. Activities must be curtailed and a regimen outlined because a permanent injury has been done to the heart. Of greater importance is the fact that, in most instances, the coronary arteries outside the area of the healed infarct are sclerotic in varying degrees. On the other hand, many of these persons are active men, engaged in carrying on important affairs, who are eager to return to work. To do justice to both the individual and his disability furnishes a problem worthy of serious and careful consideration. Each case must be decided according to its own peculiar circumstances, which include personal characteristics, occupation, economic status and the condition of the heart. The presence of significant hypertension calls for greater conservatism than when the pressure is normal. The guiding principle should be to allow as much freedom of activity as is consistent with safety and good judgment. This, to be sure, is an indefinite suggestion; but specific advice rests upon so many varying factors that generalizations are neither feasible nor wise. Happily, in many instances, patients are able to do more, without discomfort or apparent harm, than at first appears possible.

### The Moderate Type, Without Shock

*Case 3.* A male executive, aged forty-seven years, had had twinges of substernal pain for many years, but more marked for the few weeks preceding the onset of the present illness. He was a heavy cigarette

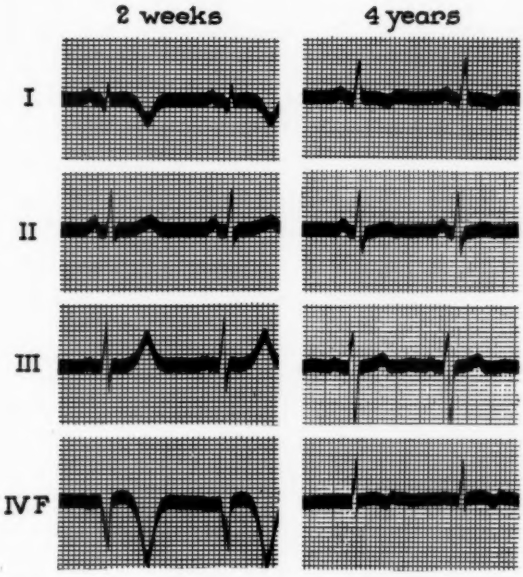


**Fig. 3. Electrocardiograms of Case 8 after the acute attack, taken at the intervals indicated.**

smoker. An electrocardiogram taken six months before the acute upset was normal, and he had been told that his discomfort was due to tobacco poisoning. The onset was acute, in the early morning. The pain was only of moderate severity and was not followed by shock. The electrocardiogram was typical of anterior infarction (Fig. 3). The leukocyte count was 20,000 with 88 per cent polymorphonuclears. The sedimentation rate, which reached a peak of 80 mm. in one hour on the seventh day, returned to normal at the end of the sixth week. He was kept in bed for seven weeks. There was no further discomfort in the chest after the initial pain had been controlled by morphine, and convalescence was uneventful. He was allowed to take a few steps on the fourth day out of bed. He did not attempt any work for four months and began by short sessions at his desk, at home. During the second summer, eighteen months after the attack, he was permitted to play golf on a level course, and did so without apparent injury to the heart. He started with a few holes and gradually worked up to eighteen. He has taken two drinks of alcohol daily.

It is now four years since the attack and he is working actively. He still rests for an hour after luncheon. After having gotten on without cigarettes for six months, the craving became too strong and he resumed his old habit at the rate of twenty or more each day.

The question of smoking almost invariably comes up for discussion. If the patient can give up tobacco entirely without hardship, it seems probable, though it is only fair to add, unproved, that he will fare better. On the other hand, many of these individuals derive a good deal of pleasure and emotional stability from smoking in moderation. By moderation, is meant from six to ten cigarettes a day. Two or three thin, mild cigars or three or four pipefuls of tobacco may be considered the equivalent of the given quota of cigarettes. The action of tobacco on the circulation appears to be a highly individualized matter. Some persons are susceptible to its effects, whereas others smoke freely without demonstrable harm. If a patient notices cardiac pain, palpitation or extrasystoles after smoking, it is well for him to stop entirely or cut down materially. Occasionally the "denicotined" brands appear to be tolerated better; but it is doubtful whether such slight differences in nicotine content are important.

Coffee, except in sensitive subjects, does no harm. Sometimes it causes premature beats, tachycardia or even cardiac pain, and should then be discontinued.[7] It may be replaced by Sanka. It is well to remember that a cup of strong tea contains about as much caffeine as the average cup of coffee.

No matter how complete the degree of recovery, it is unwise to permit indulgence in violent sports, such as squash, tennis, vigorous gymnastic exercises or swimming for long distances. Ideal forms of recreational activity are walking, golfing on a level course or horseback riding. Swimming slowly and not too far is likewise permitted; but to fight breakers in a heavy surf is inadvisable. When exercise is limited because of occupation or other circumstances, light, general massage two or three times a week serves to maintain tone in the muscles and peripheral circulation.

If attacks of anginal pain appear with the resumption of activity, nitroglycerine should be taken freely, not only for relief but to prevent discomfort when some necessary activity or stress is anticipated. To prevent nocturnal attacks, erythroltetranitrate or mannitol hexanitrate, ½ grain tablet taken just before retiring, will often aid in securing un-

disturbed sleep. The prolonged vasodilator action of these drugs is especially helpful in the presence of hypertension. The value of the xanthine derivatives as coronary dilators, when given by mouth, is still disputed. Observations made in our laboratory on patients indicate that administration of one of these drugs is clearly helpful in certain cases.[8,9] Our studies have been made chiefly with aminophyllin, given in a dose of 3 grains, three or four times daily. Sometimes smaller amounts are effectual. Administration should be continued over a period of months if painful seizures become fewer and less severe. It is difficult to select favorable subjects in advance and clinical trial is usually required. It seems likely that the drug acts largely by preventing or relieving coronary spasm; hence it is in the cases with less advanced lesions that the best results may be expected. The combination with aminophyllin of phenobarbital and a member of the atropine series, sometimes serves more effectively than any one of these alone.

In my hands, papaverine, given by mouth, even in large doses, has been worthless. The same may be said of the various organ and tissue extracts, whether given orally or by injection.

There is a small group of patients who suffer so intensely from anginal pain that some form of surgical therapy is justified. This is not the place for a consideration of the various procedures which have been employed. Accumulated experience warrants the conclusion that paravertebral sympathetic block with alcohol is the safest of these measures; when done by a skilled operator, it is effective in about 75 per cent of cases.[10]

Should it become necessary to perform a major surgical operation, such as cholecystectomy, the patient who has recovered from cardiac infarction stands it surprisingly well. If he is subject to congestive failure, the risk is clearly increased. After a preliminary dose of avertin, inhalation anesthesia may be begun with gas and oxygen, with emphasis to the anesthetist on plenty of oxygen. The operation is then continued under the influence of ether. When the margin of cardiac or coronary reserve is small, the postoperative use of an oxygen tent is helpful.

May the patient with a healed cardiac infarct fly or take up his residence at a high altitude? Provided there have been no symptoms or signs of congestive failure and there are no attacks of anginal pain or these are induced only after severe exertion, the answer is in the affirmative. During the ordinary aeroplane flight a height of 12,000 or

13,000 feet is rarely exceeded and the degree of anoxia produced at this altitude appears to be well borne. For example, a man of forty-six, who had his attack of coronary occlusion eight months ago, recently flew to Mexico City, remained there at an altitude of 7800 feet for two weeks and flew back to New York. During the flight the maximal altitude attained was 13,000 feet. He was cautioned against strenuous exertion while in Mexico. The transaction of business there tired him more readily than usual but he experienced no pain or dyspnea and there were no later ill effects.

Another patient, at the age of seventy-one, had a mild attack due to occlusion of an anterior branch, and made a splendid recovery. Three years later, long after the form of the electrocardiogram had returned to normal, he still noted a feeling of tightness in his chest on walking rapidly, but discomfort was not of sufficient severity to require the use of nitroglycerine. He was eager to spend the summer on his Western ranch at an altitude of 9200 feet and, with some trepidation on my part, permission to do so was given. While there, he walked and rode horseback without any unpleasant sensation in his chest. For the past four summers this experience has been repeated. Now, at the age of seventy-seven, he is convinced that he feels better in the mountains of Colorado than on the streets of New York.

*Case 4.* This case is included in order to indicate how failure to enforce adequate rest during the early stages may be followed by disastrous consequences. A forty-four year old liquor salesman for some years had noticed dyspnea on running up a flight of stairs. There had been no cardiac pain. Four weeks before his admission to the hospital, he developed a heavy feeling in his chest and a few hours later, during the night, experienced a severe pain beneath the sternum, radiating down the left arm. His physician gave him a sedative by hypodermic injection. Four days later he had a second, similar attack. He was kept in bed for ten days and then was allowed to get up and go about. During the next fortnight he became short of breath and had anginal pain radiating to the left arm.

He was admitted to the hospital exactly four weeks after his initial attack. The electrocardiogram was characteristic of an anterolateral infarct. There were signs also of a small pulmonary infarct in the left lower lobe. The sedimentation rate was elevated, but the leukocyte count was normal. There was slight fever for the first week, up to

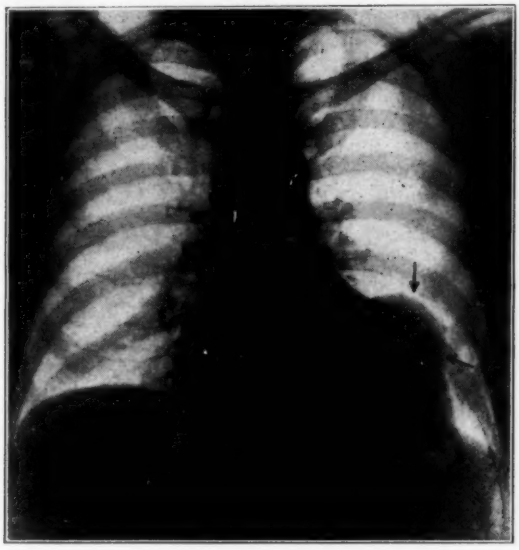

Fig. 4. Teleroentgenogram of Case 4. The arrows point to the cardiac aneurysm, which was first photographed four weeks after the occurrence of acute coronary occlusion.

100.4° F. The blood pressure was 100/70. It fell gradually to 80/50. He experienced a good deal of pain in the left shoulder. An x-ray film of the heart showed a large bulge in the border of the left ventricle, which subsequent roentgenological examinations, both fluoroscopic and kymographic, proved to be a cardiac aneurysm (Fig. 4).

This patient has been followed in the clinic for eight years. He has never been able to resume work. He has suffered from severe anginal pain and, at times, from bouts of congestive failure. There have been two subsequent attacks of coronary occlusion with signs of cardiac infarction, both mild, but requiring care in the hospital. The appearance of the aneurysm has remained essentially unchanged.

*Discussion.* A cardiac aneurysm may develop even when the initial rest period has been carefully supervised. But any added effort made while the infarct is soft will tend to aggravate such a tendency. There is experimental evidence to support this statement.[11] In normal dogs, cardiac infarction was produced by ligation of a coronary artery. In those animals allowed to rest for six days before being exercised on a

motor-driven treadmill, a small, firm scar was produced. In others, made to run sooner, a thin, bulging area was observed in the affected portion of the ventricle. The rate of healing in the myocardium of the healthy dog is clearly faster than in the diseased human heart.

It is logical to enforce complete inactivity long enough to let edema about the lesion subside, to permit absorption of the necrotic muscle and its replacement by scar tissue, and to encourage the development of a collateral circulation. The crucial period is during the early weeks and no amount of curtailment of activity later can compensate for rest which is lost at a time when it counts most. The patient who is comfortable at the end of a week or two often finds it difficult to understand why he is urged to lie so quietly and is still fed by the nurse. Attention should be focussed on his future welfare and he must be induced to sacrifice present desires for a chance to recover as completely as possible. There can be no compromise; for in restraint during this early period lies the core of successful management.

In the case described the duration of rest was only two weeks and the aneurysm of the ventricle was seen in the x-ray film four weeks after the acute occlusion. Although kept in bed for four weeks after admission to the hospital, the subsequent course made it clear that the damage already done was irreparable. Some patients with a thin and bulging ventricular wall are surprisingly well and active, considering the nature of their disease. Others, like this man, are incapacitated and uncomfortable, although they survive for a number of years.[12]

### The Mild Type

*Case 5.* A retired lawyer, seventy years of age, for two years had experienced shortness of breath on effort. One evening he began to retch and vomit, and felt a sense of constriction in the lower part of the chest. This sensation lasted three or four hours and was accompanied by profuse sweating. The next day he felt weak, but comfortable. Three days later, he went to another city to consult a gastroenterologist, who had been treating him for a number of years for digestive disturbances. He was given a mixture of antacids and sent home. On climbing the stairs at the Pennsylvania Station at the termination of his return trip, he was short of breath and had pain in his chest. He remained at home for a few days and then attended to his affairs downtown. He was seen three weeks after the onset, because he wanted a check-up before going to the country for the summer.

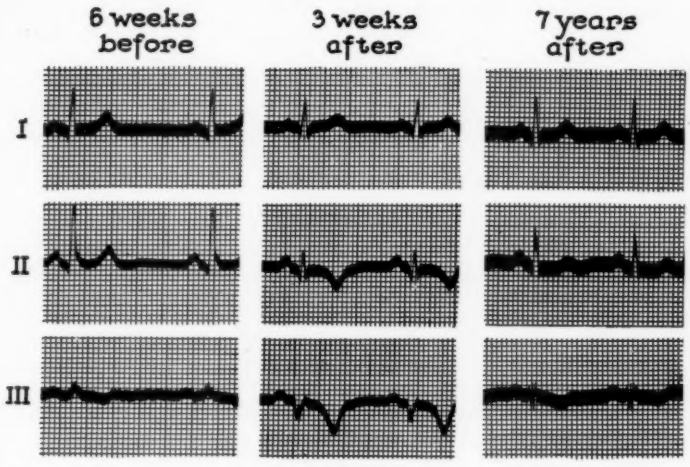


Fig. 5. Electrocardiograms of Case 5 before the acute attack and after its occurrence, taken at the intervals indicated.

The heart was not enlarged. The first sound at the apex was very weak. The blood pressure was 114/62. The electrocardiogram was typical of posterior infarction (Fig. 5). It would have been difficult at this point to persuade the patient to go to bed for he was perfectly comfortable and could hardly believe that his heart was affected. He was permitted to go to the country by motor on the following day, accompanied by a nurse. He was carried to his room on the second floor and remained on that floor for four weeks. He arose late in the morning and rested for two hours each afternoon. The prescribed month of rest was taken with equanimity. Resumption of a more active life was very gradual. No cardiac medication was prescribed.

These events took place more than seven years ago and the patient is now seventy-seven years of age. He still takes part in various civic and philanthropic affairs. There is no cardiac discomfort. The electrocardiogram shows minor residual changes.

*Discussion.* In this instance, coronary occlusion occurred in an elderly man without hypertension and with a heart of normal size, who for two years had had symptoms due to coronary insufficiency. In all probability, an adequate collateral circulation had developed so that infarction was followed by minimal symptoms and only slight functional derangement of the heart. For three weeks the nature of the illness

was unrecognized and he might well have recovered completely without ever knowing that the upset was cardiac and not digestive in origin.

If such a case is seen immediately after the onset, a period of rest in bed for three or four weeks usually suffices. Fever lasts only a few days and is not high. The leukocyte count may be only nine or ten thousand; and the sedimentation rate returns to normal within two or three weeks. In contrast to these slight disturbances, the changes in the form of the electrocardiogram are relatively of greater magnitude. The degree of distortion of the complexes is not a guide as to the severity of the lesion or the prognosis. Minimal alterations may be observed shortly before sudden death; bizarre and greatly distorted sequences occur in the presence of a benign clinical picture. The electrocardiogram may be expected to show change in serial records, often in the direction of normal. But such change is not to be used as an important aid in management, for it may continue for weeks or months, and even for years. A sudden shift in form suggesting an advancing lesion, even in the absence of symptoms, is a signal for caution and watchfulness. The course of action will depend upon the appearance of other signs.

*Case 6.* Not all of the mild cases are as fortunate as the one whose history has just been given. A stock broker, aged fifty-eight, had been a remarkably healthy man. His father and one brother died of heart disease. While in the south playing golf, he experienced substernal oppression and indigestion. He continued to play and had four seizures on the course. He was finally taken home in an ambulance. The pain was not severe, but he felt limp. He remained in bed for five days and then was taken to a hospital, where an electrocardiogram was made which, according to the report, showed inversion of $T_1$ and $T_2$ and was interpreted as indicating coronary occlusion. He remained in bed for a week and then came home to New York, by train. Because he felt perfectly well he went to his office. Nocturnal oppression and indigestion recurred. Three weeks after the onset he had a sense of pressure which lasted all day; and it was four days later that I saw him.

On examination, the heart was not enlarged. The sounds had a thin, sharp quality. The blood pressure was 124/82. The electrocardiogram showed the T waves in Leads I and II to be of low amplitude and diphasic. Although it was almost four weeks after his initial symptoms, he was having attacks of prolonged substernal oppression, indicating that the pathological process in the heart was still active. He was advised

to go home to bed under the care of nurses and this recommendation was made in a letter to his physician. The patient refused and continued to attend to his business downtown. At the end of two weeks, he called on the telephone to poke fun at the diagnosis and laugh at the advice which had been given. Ten days later, and seven weeks after the first appearance of discomfort, his obituary appeared in the morning paper under the headline, "Retired Broker Dies Unexpectedly at His Summer Home. Heart Disease Is Fatal."

*Discussion.* When the diagnosis of cardiac infarction has been made on adequate evidence and symptoms persist in the form of frequently recurring pain or paroxysmal dyspnea, the patient should be put to bed. A major occlusion cannot always be prevented, for even at rest, the occlusive process may extend. But under stress, involvement of only a small branch can cause acute coronary insufficiency and initiate ventricular fibrillation; it is probable that such a mechanism was responsible for sudden death in this instance.

## Comment

In the title of this paper it has been stated, with intent, that consideration would be given to the management of the patient; no mention was made of the treatment of his disease. Impairment or stoppage of the flow of blood through a coronary artery causes injury to that portion of the myocardium which receives its nutrition from the affected vessel. The processes of repair which follow are the reactions of the tissues to that injury. They are adaptive reactions designed to promote healing of the lesion and provide new channels of circulation in its vicinity. Unfortunately, they do not always accomplish this end. There is no form of medicinal therapy which directly modifies the structural changes which take place. Surgical procedures designed to form a new blood supply to the heart, such as the transplantation of a pectoral muscle or the injection of foreign substances into the pericardial sac, have not yet proved either their safety or effectiveness. The chief aims of the physician, therefore, are to safeguard the heart during its convalescence by lessening its work and to aid in maintaining its adequate functional performance with drugs, when this may be necessary. After recovery from the acute upset the cardiovascular system must be protected, insofar as possible, from further injury and strain. Often, much that is helpful can thus be accomplished; but because the basic influ-

ences which modify the biology of atherosclerosis are at present beyond control, the disease progresses at its own rate and in its own way.

In his presidential address before the Congress of American Physicians and Surgeons in 1897, Dr. William H. Welch discussed "Adaptation in Pathological Processes."[13] Both in content and form the paper is a classic, and forty years later was reprinted as a booklet by the Institute of the History of Medicine of the Johns Hopkins University. It is with quotation of the final paragraph that I shall conclude.

"The healing power of nature is, under the circumstances present in disease, frequently incomplete and imperfect, and systems of treatment based exclusively upon the idea that nature is doing the best thing possible to bring about recovery or some suitable adjustment, and should not be interfered with, rest often upon an insecure foundation. The agencies employed by nature may be all that can be desired; they may, however, be inadequate, even helpless, and their operation may add to existing disorder. There is ample scope for the beneficent work of the physician and surgeon."

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## PROCEEDINGS OF ACADEMY MEETINGS

### *STATED MEETINGS*

OCTOBER 1—*The New York Academy of Medicine.* ¶ Executive session. Reading of the Minutes. ¶ Papers of the evening—Scientific program under the joint sponsorship of The New York Academy of Medicine and its Section of Surgery and Section of Ophthalmology. ¶ Chemical Warfare as Affecting Civilian Population—a] Pulmonary irritants, Robert A. Kehoe, Research Professor of Physiology, College of Medicine, University of Cincinnati; b] Vesicants, Leon Goldman, Assistant Professor of Dermatology, College of Medicine, University of Cincinnati; c] The effect of irritating and destructive gases and chemicals on the eye, Conrad Berens, Professor of Ophthalmology, New York University College of Medicine; Edward Hartmann, Formerly Chief of the Ophthalmologic Service, American Hospital of Paris, France. ¶ Report on Election of Fellows and Corresponding Fellows.

OCTOBER 29—*The Harvey Society in affiliation with The New York Academy of Medicine.* ¶ The First Harvey Lecture —"The Significance of Labile Methyl Groups in the Diet and Their Relation

to Transmethylation." Vincent Du Vigneaud, Professor of Biochemistry, Cornell University Medical College.

NOVEMBER 5—*The New York Academy of Medicine.* ¶ Executive session: 8:30 o'clock. a] Reading of the minutes; b] Report of Nominating Committee. ¶ Papers of the evening: Scientific program under the joint sponsorship of The New York Academy of Medicine and its Section of Medicine. ¶ Special Therapeutic Agents in Infections. a] Gramicidin, René J. Dubos, George Fabyan Professor of Comparative Pathology and Tropical Medicine, Harvard University; b] Clinical application of gramicidin and penicillin, Chester S. Keefer, Wade Professor of Medicine, Boston University School of Medicine; c] Sulfonamide therapy in diseases of the intestinal tract, Warfield M. Firor, Visiting Surgeon, Johns Hopkins Hospital. ¶ Report on Election of Fellows.

NOVEMBER 19—*The Harvey Society in affiliation with The New York Academy of Medicine.* ¶ The Second Harvey Lecture, "Total Homeostasis," Curt P. Richter, Associate Professor of Psycho-Biology, Johns Hopkins University.

## *SECTION MEETINGS*

OCTOBER 6—*Dermatology and Syphilology.* ¶ Presentation of cases. ¶ Discussion. ¶ Executive session: Nomination and election of secretary.

*Surgery*—This Section held no meeting on its regular date as it combined with the State Meeting of October 1.

OCTOBER 6—*Combined Meeting Neurology and Psychiatry and the New York Neurological Society.* ¶ Presidential address, Neuropsychiatry in War Time, G. A. Blakeslee. ¶ Papers of the evening—a] Early laminectomy in spinal cord injuries, F. Kennedy, P. G. Denker, R. L. Osborne; discussion by E. J. King, Byron Stookey; b] Effect of Vitamin E therapy on the central nervous system in amyotrophic lateral sclerosis, Charles Davison; discussion by Tracy J. Putnam, I. S. Wechsler.

OCTOBER 8—*Pediatrics.* Reading of the minutes. ¶ Presentation of cases: Program arranged by Pediatric Service of Mt. Sinai Hospital—a] An acute febrile illness with rash and leukopenia due to H. para-influenza, Alfred R. Florman (by invitation); discussion by Hattie Alexander (by invitation); b] A comparison between protamin and regular insulin in the young diabetic, Alfred E. Fischer, discussion by Frederick W. Williams; c] Idiosyncrasy to mercury in childhood, Murray H. Bass; d] The pathological kidney findings as compared with the clinical course of lipoid nephrosis in childhood, Jerome Kohn; discussion by Herman Schwarz, John D. Lyttle; e] Management of pulmonary abscess in childhood, George Ginandes (by invitation); discussion by Harold Neuhof. ¶ General discussion. ¶ Executive session.

*Ophthalmology*—This Section held no meeting on its regular date but combined with the Stated Meeting of October 1.

OCTOBER 27—*Obstetrics and Gynecology.* ¶ Executive session—a] Reading of the minutes; b] Nomination and election of secretary. ¶ Papers of the evening—a] Syphilis in pregnancy—End-results in treatment, Mortimer Dudley Speiser; b] Highlights in the diagnosis and treatment of gonorrhea in women (Public Health viewpoint), Adolph Jacoby; c] Gonorrhea—diagnosis and treatment, R. Gordon Douglas (by invitation). ¶ General discussion—Opened by David Nye Barrows and Louis Chargin—The Section regrets the resignation of Locke L. Mackenzie as Secretary due to his entering the navy.

*Genito-Urinary Surgery—Medicine—Orthopedic Surgery—Otolaryngology*—These Sections held no meeting in October be-

cause of conflict with meeting dates of the Graduate Fortnight.

NOVEMBER 4—*Dermatology and Syphilology.* ¶ Presentation of cases—a] From the Skin and Cancer Unit of the Post-Graduate Medical School; b] Miscellaneous cases. ¶ Discussion. ¶ Executive session.

NOVEMBER 4—*Historical and Cultural Medicine.* Reading of the minutes. ¶ Papers of the evening—Public Health in the City of New York—a] Introduction, by James Alexander Miller; b] Retrospect, by E. H. L. Corwin and Charles Bolduan; c] Glimpses of the future, by Ernest L. Stebbins (by invitation). ¶ General discussion.

NOVEMBER 6—*Surgery.* Reading of the minutes. ¶ Case presentation — Unusually large adenoma of the adrenal gland, Francis X. Timoney. ¶ Case report—Congenital absence of common bile duct, Stanley J. Brady (by invitation). ¶ Papers of the evening—a] An eight-year review of ruptured peptic ulcers at St. Vincents' Hospital, with special reference to the local use of sulfonamides, Francis X. Timoney; discussion by Constantine MacGuire; b] Reconstruction of the common bile duct, Raymond P. Sullivan. ¶General discussion. ¶ Executive session.

NOVEMBER 10—*Combined meeting Neurology and Psychiatry and the New York Neurological Society.* ¶ Papers of the evening—a] The selective use of electro-shock therapy as an adjuvant to psychotherapy, Herman Selinski; discussion by Bernard B. Glueck and R. B. McGraw; b] Primary diffuse sarcomatosis of meninges, M. P. Rosenblum (by invitation), and Lewis D. Stevenson; discussion by Joseph H. Globus; c] Limitations of psychoanalytic therapy, Herman Nunberg; discussion by A. A. Brill and Bertram D. Lewin.

NOVEMBER 12—*Pediatrics.* Reading of the minutes. ¶ Round Table Discussion—Eczema in infants and children, Robert A. Cooke, Marion B. Sulzberger, Anthony C. Cipollaro and Howard H. Mason. ¶ General discussion.

NOVEMBER 16—*Ophthalmology.* ¶ Instruction Hour: 7.00 to 8.00 o'clock—War injuries of the eye, Edward Hartman (by invitation). ¶ Executive session—Reading of the minutes. ¶ Presentation of cases—a] Bilateral metastatic carcinoma of the choroid, Arthur J. Bedell; discussion by Arnold H. Knapp; b] Neurodermatitis with cataract, Clyde McDannald; discussion by Truman L. Boyes. ¶ Papers of the evening—a] Immediate and late reoperation for glaucoma, Willis S. Knighton; b] Histologic causes for failures in glaucoma operations, Brittain F. Payne; discussion by A. B. Reese.

*Section of Medicine*—The Section of Medicine did not hold its regular meeting on November 17, as it combined its scientific program with that of the Stated Meeting on November 5.

NOVEMBER 18 — *Genito-Urinary Surgery.* Reading of the minutes. ¶ Case reports —a] Enormous prostatic diverticulum, E. King Morgan; b] Diverticulum of posterior urethra with stones, O. P. Schoenemann; c] Reflex renal shutdown after prostatic resection, T. A. Morrissey (by invitation). ¶ Papers of the evening—a] Prevention of renal obstruction on massive sulfadiazine therapy, Ole Jensen (by invitation); b] Further experiences with vitallium tubes in the urinary tract of the dog, Jere Lord (by invitation); c] Hyperplastic change at the vesical neck in the female, Joseph A. Hyams and Sidney Weinberg (by invitation). ¶ General discussion. ¶ Executive session.

NOVEMBER 18—*Otolaryngology.* Reading of the minutes. ¶ Papers of the evening—a] Granular cell myoblastomas of the external auditory meatus, Franz Altmann (by invitation); b] Principles of

diagnosis and treatment of allergy as related to otolaryngology, French K. Hansel, St. Louis, Mo. (by invitation); discussion by Marvin Jones and R. C. Grove. ¶ General discussion. ¶ Executive session.

NOVEMBER 20—*Orthopedic Surgery.* Reading of the minutes. ¶ Presentation of cases—a] A case of disability of the knee joint, Alvin Hulnick (by invitation). ¶ Papers of the evening—a] Recurrent dislocation of the patella, Walter Thompson (by invitation); discussion by Lewis Clark Wagner; b] The repair of the ruptured crucial ligaments of the knee, Fred H. Albee; c] "Hey-Groves' Operation for Repair of Crucial Ligaments"—Motion picture and discussion of the subject, Arthur Krida. ¶ General discussion. ¶Executive session.

NOVEMBER 24—*Obstetrics and Gynecology.* ¶ Executive session — Reading of the minutes. ¶ Case report — Pregnancy complicated by a large myoma, Arthur F. Wright (by invitation). ¶ Round table discussion—The menopause and its problems, Frederick C. Holden, Chairman, Harvey B. Matthews, Samuel H. Geist, and Ralph L. Barrett. ¶ General discussion.

## *AFFILIATED SOCIETIES*

OCTOBER 29—*New York Pathological Society in affiliation with The New York Academy of Medicine.* ¶ Papers of the evening—Medically unimportant autopsy findings which are likely to be of paramount interest from a medico-legal standpoint, Alan R. Moritz (by invitation). ¶ Executive session—A vote was taken on the proposed revision of the Constitution.

*New York Roentgen Society in affiliation with The New York Academy of Medicine.* Because of conflict in dates with the Graduate Fortnight, this Society held no meeting in October.

NOVEMBER 12—*New York Pathological Society in affiliation with The New York Academy of Medicine.* ¶ Presentation of cases—a] Meningioma with invasion of the pituitary gland, John J. Larkin (by invitation); b] Agenesis of lung—Report of case, Charles T. Olcott; c] Acute febrile anemia in thrombocytopenia with generalized platelet thrombi, Paul Klemperer. ¶ Paper of the evening—The mechanism of jaundice in carcinoma of the pancreas, Naomi Kaplan (by invitation) and Alfred Angrist. ¶ Executive session.

NOVEMBER 16—*New York Roentgen Society in affiliation with The New York Academy of Medicine.* ¶ Papers of the evening—a] Non-luetic aortic aneurysms, M. F. Steinberg; b] Atypical coarctation of the aorta, A. Grishman; c] Roentgen appearance of right heart enlargement, M. L. Sussman; discussion by A. H. Blakemore (by invitation) and Ernst Boas (by invitation). ¶ Executive session.

## *DEATHS OF FELLOWS*

ALLEN, THEOPHILUS POWELL: 16 East 90 Street, New York City; born in Milledgeville, Georgia, July 24, 1896; died in New York City, January 27, 1943; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1923; elected a Fellow of the Academy November 6, 1930.

Dr. Allen was associate attending physician to St. Luke's Hospital; assistant attending physician to the Neurological and Presbyterian Hospitals; a Fellow of the American Medical Association, and a member of the National Gastroenterological Association, and the State and County Medical Societies.

BOLDT, HERMANN JOHANNES: White Plains, New York; born in Neuentempel, near Berlin, Germany, June 24, 1856; died in St. Petersburg, Florida, January 12, 1943; graduated in medicine from New York University of the City of New York Medical Department in 1879; elected a Fellow of the Academy June 5, 1884.

Dr. Boldt was emeritus professor of gynecology at the New York Post-Graduate Medical School, where he had been professor of gynecology from 1891 to 1917; consulting gynecologist to the New York Post-Graduate Medical School and Hospital, Stuyvesant, Polyclinic, St. Vincent's, Beth Israel and Union Hospitals. He was one of the founders, formerly a member of the Board of Governors, and Fellow of the American College of Surgeons, a member of the Southern Surgical Society, an honorary member of the American Gynecological Society, and Gynecological Society of Great Britain, a member of the National Society of Sciences, the New York Academy of Sciences, the Association of Military Surgeons, and the Royal Society of Medicine, London.

Dr. Boldt was the inventor of an operating table for abdominal surgery which won a medal at the Paris exposition in 1900, and was known as an extensive investigator on the physiologic action of cocaine and gynecologic pathology.

MORRIS, JOHN HAROLD: 535 Park Avenue, New York City; born in Cortland, New York, July 29, 1889; died in New York City, November 30, 1942; graduated in medicine from Cornell University Medical College in 1914; elected a Fellow of the Academy January 3, 1924.

Dr. Morris was associate clinical professor of surgery at New York University College of Medicine and visiting surgeon to the Bellevue Hospital, St. Vincent's Hospital and the Goldwater Memorial Hospital at Welfare Island, formerly the Hospital for Chronic Diseases. He was a diplomate of the American Board of Surgery, a Fellow of the American College of Surgeons, a Fellow of the American Medical Association, a member of the New York Surgical Society, and a member of the State and County Medical Societies.

SCHILLER, ABRAHAM NOAH: 27 West 72 Street, New York City; born in New York City, April 15, 1884; died in New York City, December 17, 1942; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1907; elected a Fellow of the Academy May 3, 1923.

Dr. Schiller, one of the founders of the Jewish Memorial Hospital, was honorary chairman and chairman of the medical board, consulting otolaryngologist and for many years chief of the department of otolaryngology at that institution; consulting otolaryngologist and attending physician in the ear, nose and throat division of the New York City Hospital, Welfare Island; a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association and the State and County Medical Societies.